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THE DEVELOPMENT AND ANOMALIES OF THE AORTIC ARCH AND ITS BRANCHES

WITH THE REPORT OF A CASE OF RIGHT CERVICAL AORTIC ARCH AND INTRATHORACIC VASCULAR RING

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THE following case was reported in detail up to 1953 by Lewis and Rogers (1953), and a summary only of their findings will be given here. Since that time the patient has developed dysphagia, caused by a vascular ring surrounding the trachea and œsophagus in the chest.

CASE REPORT

In 1937, when a boy of 5 years of age, the patient was investigated in the children's department of the Cardiff Royal Infirmary (Dr. A. G. Watkins), as a case of aneurysm of the right common carotid artery. A pulsating swelling had been noticed in the lower part of the right side of the neck for three years, and had not altered appreciably during that time. He was a delicate boy who always wanted to rest, he became short of breath on walking to school, and his lips became blue at times. In the region of the right common carotid artery there was an oval pulsating swelling, measuring 1½ in. in its long axis, over which was a thrill and a bruit. On the left side axillary pulsations were faint, while the brachial, radial, and ulnar pulses were absent. Pulsation in the right arm and over both femoral arteries was normal. The blood-pressure in the right arm was 95/64 mm. Hg. The cardiovascular system was otherwise normal, the electrocardiogram was normal, and radiography showed minimal enlargement of the heart and slight widening of the upper mediastinal shadow.

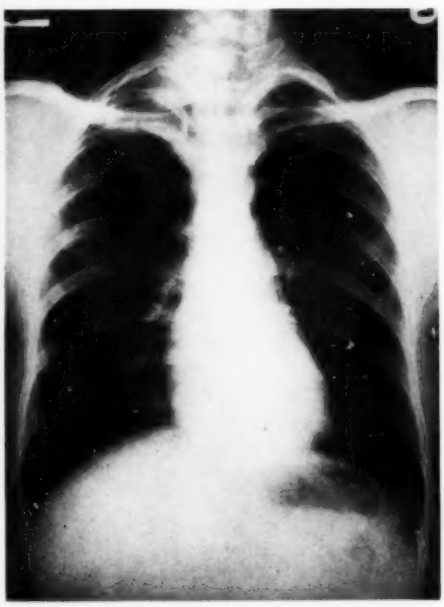


FIG. 635.—Radiogram of chest, showing opacity at right apex.

FIG. 636.—Penetrating radiograph of neck, showing narrowing of upper third of trachea.

He was readmitted to the Cardiff Royal Infirmary in 1939 under Professor Lambert Rogers, complaining of dyspnœa and cyanosis on exertion, and of diplopia of two years' duration. Operation in August, 1939, on his neck revealed the presence of a right-sided aortic arch reaching

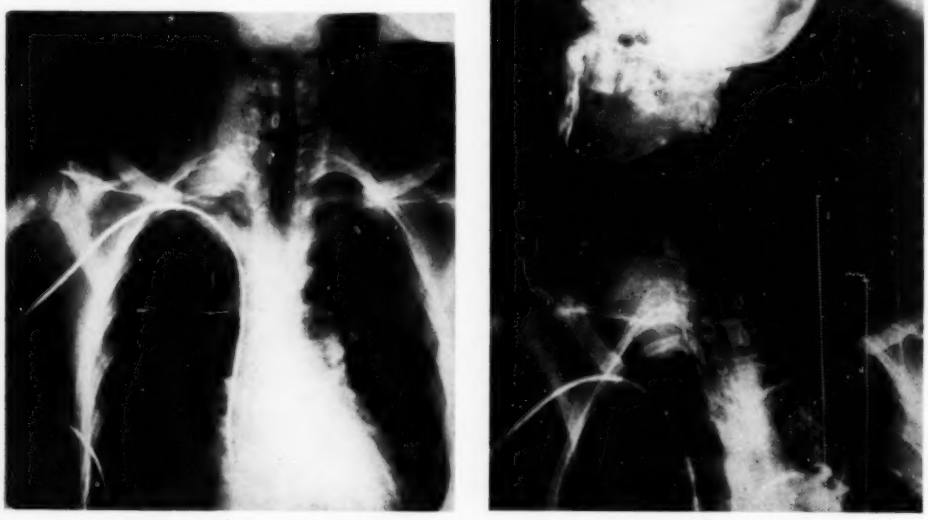
to the level of the hyoid bone, where it formed an acute anteroposterior arch. The afferent limb lay in front of and slightly medial to the efferent limb of the arch, from the summit of which arose the right internal and external carotid arteries (*Fig.* 639). The left common carotid artery arose from the ascending limb and the right subclavian artery from the descending limb of the aortic arch. In January, 1940, Professor Lambert Rogers reinforced the area over the cervical aortic arch with a graft of fascia lata. At this operation it was noted that compression of the vessel stopped the femoral pulses.

The patient was readmitted in May, 1950, because of two fainting attacks while at work, and headaches after reading. The cervical swelling measured 3 in. long by 1½ in. wide, the trachea was displaced to the left, the left radial pulse was present, but weaker than the right, the blood-pressure in the right and left arms was 112/86 and 92/84 mm. Hg respectively, and the right palpebral fissure was narrower than the left. The temperature in the right hand was 5° higher than in the left hand. Barium swallow showed a smooth indentation of the posterior wall of the œsophagus opposite the fourth and fifth thoracic vertebrae. Angiography was unsuccessful. Electro-encephalography showed scattered low-voltage theta waves in all leads, intermingled with alpha rhythm, but hyperventilation had not much effect on the record, which was considered to be outside the normal range.

He was again readmitted under Professor Lambert Rogers in March, 1957, with a history of difficulty in swallowing potatoes and meat for six months. In order to get these substances down he had to swallow twice and take a drink of water. The dyspnœa and cyanosis from which he used to suffer had completely disappeared, and he had had no further fainting attacks since his last admission. The swelling in the neck had been gradually increasing in size over the years. He was referred to me because of the dysphagia.

On examination there was a pulsating swelling in front of the right sternomastoid muscle, reaching from the clavicle to the angle of the mandible, over which there was a systolic murmur. The swelling exhibited two pulsations to each heart-beat, and the pulsation caused movement of the entire larynx. Pulsation on the left side of the neck and in both supraclavicular fossæ was normal. The right radial pulse was normal, but the left was distinctly smaller. The blood-pressure in the right arm was 140/110 mm. Hg. Both femoral pulses were present and equal. The cardiovascular system was otherwise

FIG. 637.—Barium swallow, showing indentation of posterior wall of œsophagus at level of manubriosternal junction.



A B

FIG. 638.—A, B, Venous angiocardiograms showing aortic arch in right side of neck with tortuous descending limb, the left innominate artery, and the right subclavian artery.

normal except for accentuation of the aortic second sound, and electrocardiography showed no abnormality. A radiograph of the chest showed slight prominence in the region of the pulmonary artery, absence of the aortic knuckle, and a uniform, well-defined opacity at the apex of the right hemithorax, the outer margin of which coincided closely with the inner margin of the first rib (*Fig.* 635). The trachea was displaced to the left. No enlargement of the heart or any of its chambers was found on screening, and the diaphragms moved normally. Penetrating radiographs of the neck (postero-anterior and lateral) showed that the trachea was displaced to the left and narrowed in its upper one-third, but normal in its lower two-thirds (*Fig.* 636). Barium swallow showed a well-marked, smooth indentation of the posterior wall of the œsophagus at the level of the manubriosternal junction (*Fig.* 637). The indentation was transversely placed. Venous angiocardiography (*Fig.* 638 A, B) showed the aortic arch in the right side of the neck reaching up to the level of the lower part of the fourth cervical vertebra. The lateral of the two limbs (descending limb) was very tortuous and accounted for the opacity at the apex of the right hemithorax, seen on the straight radiograph of the chest. The branches of the aorta, other than the left common carotid and the right subclavian arteries, the ductus arteriosus, and the lower part of the descending aorta, were not visible.

The anatomical diagnosis was right-sided cervical aortic arch, transverse retro-œsophageal segment of aorta, left-sided descending aorta, and left-sided ligamentum arteriosum. Dysphagia was considered to be due to a vascular ring formed by the retro-œsophageal segment of aorta behind, the ligamentum arteriosum on the left side, and the division of the pulmonary artery and its left branch in front. *Fig.* 639 shows the anatomical arrangement present in this case. It was constructed from the findings at the operations upon the neck and chest, and from a study of the venous angiocardiograms.

Left posterolateral thoracotomy was performed through the bed of the fifth rib on April 2, 1957. The preoperative diagnosis was confirmed. On retracting the lung the left descending aorta and left subclavian artery were readily seen, but the arch of the aorta and its branches were not visible. Dissection revealed a retro-œsophageal segment of the aorta running horizontally from right to left and indenting the back of the œsophagus. Having crossed behind the œsophagus the aorta turned downwards at right angles to form the left-sided part of the descending aorta. The left subclavian artery arose from a conical-shaped diverticulum which ascended from the top of the angle or junction of the horizontal and descending parts of the aorta, and appeared to be an upward continuation of the latter. The apex of the cone of the diverticulum became continuous with the subclavian artery about 1 cm. above its point of continuity with the descending aorta (*Fig.* 639). The diverticulum represented a persistent part of the left paired aorta (dorsal aortic root of Arkin, 1936). A tense, obliterated ligamentum arteriosum, 2 cm. long, was attached to the left subclavian artery at its junction with the aortic diverticulum (*Fig.* 639). From here it ran to the right and somewhat forwards, at first to the left of, and then in front of, the trachea, to join the superior border of the left pulmonary artery as it joined the right pulmonary artery. The ligamentum was so tense that it angled forwards its point of attachment to the junction of the subclavian artery and aortic diverticulum, so that these structures were pulled farther forwards and inwards with each beat of the heart (*Fig.* 639). It was at first thought that the ligamentum arteriosum was attached to the aorta and that the conical narrowing, representing the beginning of the subclavian artery, was a coarctation of the aorta, but investigation proved the state of affairs described above. The recurrent nerve was given off about 5 cm. above the ligamentum arteriosum, but turned around its attachment to the left pulmonary artery in the usual way. Because of the high origin of the nerve, and its relation to the extreme right anterior end of the ligamentum, it was not demonstrated until a late stage of the operation. The left common carotid artery was felt pulsating to the right of the left subclavian artery, but was not dissected. The ascending aorta was felt farther to the right and more anteriorly.

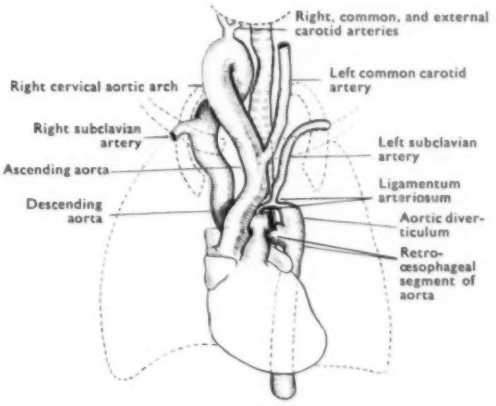


FIG. 639.—Present case, showing right-sided aortic arch in the neck, with aneurysmal dilatation and tortuous descending limb, and vascular ring in the chest. The tension in the long, left-sided ligamentum arteriosum is indicated by the angulation where it is attached to the junction of the subclavian artery with the aortic diverticulum. Note the narrowing of the trachea in the neck.

The transverse retro-œsophageal segment of the aorta, the ligamentum arteriosum, and the pulmonary arteries formed a vascular ring which fitted tightly around the trachea and œsophagus and caused a definite indentation of the posterior wall of the latter. The ligamentum was tightly applied to the left and anterior aspects of the lower end of the trachea and was situated about 1 cm. higher (i.e., nearer the head) than the retro-œsophageal segment of the aorta. The ligamentum was divided between silk ligatures and a small portion of it was taken for microscopical examination. The latter showed an obliterated artery rich in elastic tissue. The tension on the ligament was such that after division its two ends separated for 1·5 cm., the right anterior end retracting almost out of sight into the mediastinum. Constriction of the trachea and œsophagus appeared to be completely relieved. The patient has had no dysphagia in the seven months during which he has been followed up since the operation.

THE DEVELOPMENT OF THE AORTIC ARCHES

After three weeks of intra-uterine life the paired cardiopericardial plates and endocardial heart tubes fuse to form a simple, unpaired tubular heart, and the paired pericardial portions of the cœlom fuse to form a single pericardial cavity. At three and one half weeks of age atrioventricular, bulboventricular, and sino-atrial sulci appear to demarcate the bulbus cordis, primitive ventricle, primitive atrium, and sinus venosus.

The primordial endocardial tubes extend headwards beyond the pericardial region, where they fuse to form the truncus arteriosus, the pericardial attachment separating this from the bulbus cordis. At its cranial end the truncus arteriosus expands to form

the aortic sac, from which it is not separated by a line of demarcation. The aortic sac is a midline, unpaired structure which is applied to the floor of the pharynx. As these changes occur in the heart and truncus arteriosus paired aortæ form from cords and knots of cells which appear in the primitive endothelial plexus present in the mesenchyme. These strands of cells develop into endothelial tubes which are later reinforced by circularly arranged connective tissue and smooth muscle cells. The paired aortæ, at their cranial ends, are continuous with the aortic sac, and they bend laterally and ventrally around the pharynx, in the tissues of the mandibular arch. In this way the aortic sac is connected to paired dorsal aortæ by the first pair of aortic arches.

The heart of lower vertebrates becomes continuous cranially with paired ventral aortæ which communicate with paired dorsal aortæ by a series of aortic arches, as illustrated in the diagram of Rathke (1858), or one of its modifications, such as that of Barry (1951), but Congdon (1922) has shown that in man paired ventral aortæ never develop. He describes two phases of development of the aortic arch system in the human embryo, the branchial phase, which begins at 23 days of age, and which lasts for about 22 days, and the post-branchial phase, which is initiated by the interruption of the dorsal end of the right sixth aortic arch when the embryo is 12 mm. long (6 weeks). Most of the vital changes of the second phase occur during its first two weeks.

The Branchial Phase.—During this phase a series of six aortic arches develop, in the visceral arches, to connect the aortic sac with the paired dorsal aortæ. The first of these, the most headward, develops in the mandibular arch. The more caudal arches develop subsequently, and as they do so the cranial ones degenerate and disappear. Congdon (1922) dates the appearance of the arches as follows:—

First aortic arches 1·3 mm. (3 weeks).
Second aortic arches 3 mm. (3½ weeks).
Third aortic arches 4 mm. (3½ weeks).
Fourth aortic arches 4 6 mm. (4 weeks).
Fifth aortic arches—shortly after 6th arches.
Sixth aortic arches 5 mm. (4¼ weeks).

The first and second arches disappear as the third and fourth are developing. The fifth arch is ephemeral, appearing after the sixth, and disappearing almost as soon as it has formed. Each arch is formed from three elements, an outpouching from the aortic sac, a similar outpouching from the paired dorsal aorta, and an intermediate endothelial capillary plexus in the corresponding visceral arch.

As the aortic arches are developing and degenerating changes occur in the aortic sac. This is divided into aortic and pulmonary portions by deepening of the cleft between the origins of the fourth and sixth arches on each side. The pulmonary portion becomes tubular to form the pulmonary trunk, but the aortic portion remains saccular and is subsequently called the aortic sac. The latter spreads laterally to form right and left horns, which receive the third and fourth arches of the two sides. These horns correspond to the two ventral aortæ of lower vertebrates. The right horn ultimately becomes the innominate artery, and the left horn that portion of the arch of the aorta which lies between the innominate and left common carotid arteries. The aortic arches radiate from the aortic sac, and as the first and second arches degenerate while the more caudal ones develop the sac moves caudally, relative to the pharynx, so that at no time are paired ventral aortæ present.

The right and left primitive pulmonary arteries grow caudally from the aortic sac and are developed from a pre-existing capillary plexus before the sixth aortic arches are complete. At this time the lung buds are present and are largely continuous with the œsophagus, the capillary plexuses of the two being continuous, the œsophageal portion draining into the dorsal aorta. After their formation there is a marked angle where the primitive pulmonary arteries join the sixth aortic arches.

The sixth aortic arches take part in the development of the pulmonary arteries, the left fourth arch in that of the arch of the aorta, the right fourth arch in that of the right subclavian artery, and the third aortic arch of each side in the development of the common and internal carotid arteries. Further details will be given later. There is a difference of opinion regarding the development of the external carotid arteries. Congdon (1922) states that they arise as outgrowths from the aortic sac after the first and second aortic arches have disappeared. Their origin subsequently shifts to the third aortic arches, so separating the latter into a proximal part, which forms the common carotid artery, and a distal portion, which gives rise to the proximal part of the internal carotid artery. The rest of the latter vessel is formed from the paired dorsal aorta and from a headward extension of it.

The paired dorsal aortæ fuse over a considerable portion of their extent during the early part of the branchial phase of development, the process being complete when the embryo is 4–6 mm. long (about 4 weeks). Fusion begins in the mid-body region, according to Patten (1953), and spreads from there caudally throughout the length of the embryo, but cranially it stops short of the branchial region, where the aortæ remain paired. Barry (1951) points out that one can regard the dorsal aortæ as being divided into segments demarcated caudally by the corresponding dorsal intersegment arteries. According to Barry, fusion of the paired dorsal aortæ extends cranially to involve the ninth segments, but not the eighth. When the process of fusion is complete one may refer to the paired and unpaired portions of the dorsal aortæ, or simply to the paired and unpaired dorsal aortæ. Barry calls the paired aortæ the 'dorsal aortic roots', following Arkin (1936), and the right and left horns of the aortic sac the 'ventral aortic roots'. I prefer the terms 'paired dorsal aortæ' and 'right and left horns of the aortic sac' (Congdon, 1922), because they are anatomically descriptive. The term 'root' seems inappropriate to the paired aortæ.

The subclavian artery develops from the seventh dorsal intersegmental artery when the embryo is 4–6 mm. long and the anterior limb bud forms a slight elevation situated opposite to the seventh and eighth body segments. The fourth aortic arch is present, the sixth is incomplete, and the paired dorsal aortæ have fused up to the junction of the ninth and eighth segments. Each dorsal intersegmental

artery divides into a dorsal branch to the spinal cord, vertebral column, and vertebral muscles, and a ventrolateral branch to the body wall, that of the seventh artery supplying the limb bud. A longitudinal anastomosis connects the dorsal branches of the first seven arteries, ventral to the costal element of the seventh cervical vertebra, but dorsal to the costal elements of the remaining cervical vertebræ. This anastomosis becomes the vertebral artery and after its formation the rest of the first six dorsal intersegmental arteries disappear, leaving the vertebral artery attached to the subclavian artery, and entering the foramen in the transverse process of the sixth cervical vertebra. A second longitudinal anastomosis connects the ventrolateral branches of the seventh, eighth, and ninth dorsal intersegmental arteries, ventral to the costal processes. After its appearance the proximal parts of the eighth and ninth arteries disappear, leaving the anastomosis attached to the subclavian artery as the highest intercostal artery. The tenth dorsal intersegmental artery becomes the first aortic intercostal artery. The subclavian arteries, because they arise from the seventh dorsal intersegmental arteries, lie headwards of the costal processes attached to the first thoracic vertebra and are subsequently anchored in position by the first ribs, as well as by the vertebral arteries. This is important to the later development of the subclavian arteries.

By the end of the branchial phase of development the vascular arrangements are symmetrical. The aortic sac has been divided into a pulmonary portion, to which the sixth aortic arches are attached, and an aortic portion, which has developed right and left horns, to which the third and fourth arches of each side are attached. The first, second, and fifth arches have disappeared, the external carotid arteries have grown headwards from the aortic sac, the subclavian, vertebral, and highest intercostal arteries have developed, and the dorsal aortæ have fused from their caudal ends up to the level of the eighth dorsal intersegmental arteries.

The Post-branchial Phase.—Profound changes occur in the post-branchial phase which upset the earlier symmetrical arrangement. These begin at the 12-mm. stage (6 weeks) with the interruption of the dorsal portion of the right sixth aortic arch. When the embryo measures 14 mm. in length (7 weeks) the right and left paired aortæ become interrupted between the points where they receive the third and fourth aortic arches, the region known as the 'ductus caroticus', and at the 18-mm. stage (8 weeks) the eighth segment of the right paired aorta disappears. According to Congdon (1922) these events are dependent upon two factors: (1) longitudinal tension, due to caudal descent of the heart and aortæ; and (2) altered flow. Caudal descent carries the heart and aortæ from the occipital region into the thorax, and it occurs most rapidly when the embryo is increasing from 14 to 18 mm. in length (7–8 weeks). During this process the carotid and subclavian arteries become elongated. Altered flow is brought about in various ways. The division of the aortic sac into pulmonary and aortic portions occurs well to the left of the midline, so directing more blood into the left than into the right sixth aortic arch, and causing it to take a more direct route to the aorta than does its fellow of the right side. Descent of the heart causes traction on the two ends of each sixth arch against the resistance of the vagus and its recurrent branch. The more robust arch on the left side draws the vagus and its recurrent branch caudally with it, but the longer and more slender arch on the right side breaks.

The breaking of the carotid ducts on each side, according to Congdon (1922), occurs because the third aortic arch, as it joins the paired dorsal aorta, is directed cranially whereas the fourth arch is directed caudally. This creates a stagnant area between the points of attachment of the two arches which is ruptured by the tension caused by caudal descent of the heart when the embryo is 14 mm. long (7 weeks). The cranial inclination of the dorsal ends of the third arches becomes necessary, after the disappearance of the first and second arches, in order that the head end of the embryo may be supplied with blood.

During development the truncus arteriosus becomes oblique and directs more blood to the left than to the right side. In consequence of this the right fourth arch and the right paired aorta caudal to it become smaller than their counterparts on the left side. Descent of the heart and aortæ causes traction upon the eighth segments of the two paired aortæ because the subclavian arteries, derived from the seventh dorsal intersegmental arteries, are anchored by their vertebral branches and by the first ribs. Because of this, the eighth segment of the more slender right paired aorta gives way at the 18-mm. stage (8 weeks), whereas the larger left paired aorta withstands the strain.

The subsequent development of the two subclavian arteries differs. Traction upon the left subclavian artery causes its origin to shift headwards and, as described by Barry (1951), the segments of the left paired dorsal aorta cranial to this origin undergo extreme compression. These segments are the third to the seventh. According to Barry, as the heart descends the origins of the dorsal intersegmental arteries move cranially, so that the first and second arise from the region of the carotid ducts, and when these are broken the first and second segments of the paired dorsal aortæ disappear. As the third to seventh segments of the left paired dorsal aorta become compressed the eighth elongates. The shift of the left subclavian artery carries its origin headwards of the ductus arteriosus (left sixth aortic arch), and close to the origin of the left common carotid artery.

On the right side the disappearance of the ductus caroticus and of the eighth aortic segment allows the right fourth aortic arch, the third to seventh segments of the right paired dorsal aorta, and the right seventh dorsal intersegmental artery to be pulled out into a continuous slightly arched vessel, the subclavian artery.

When all the above changes have occurred and the embryo is 18 mm. long (8 weeks) the adult arrangement is achieved. We are now in a position to determine the constitution of the adult aorta and its branches. From its origin to the point where the innominate artery arises the aorta develops from the truncus arteriosus. The innominate artery arises from the right horn of the aortic sac. The left horn

of the sac gives rise to the portion of the arch of the aorta between the innominate and left common carotid arteries; the aorta between the left common carotid and subclavian arteries is derived partly from the left fourth aortic arch and partly from the third to seventh segments of the left paired dorsal aorta. On the lesser curvature the fourth aortic arch gives rise to a portion of the aorta proximal to the ductus arteriosus, which is attached to the fourth segment of the left paired dorsal aorta. The aorta distal to the left subclavian artery is developed from the eighth segment of the left paired dorsal aorta and thereafter from the unpaired aorta. The lower border of the tenth segment is indicated by the point of origin of the first aortic intercostal artery, which is developed from the tenth dorsal intersegmental artery.

The innominate artery, as described, arises from the right horn of the aortic sac. The right subclavian artery develops from the right fourth aortic arch, the third to seventh segments of the right paired dorsal aorta, and the right seventh dorsal intersegmental artery. The third arch and right paired aorta form only that part of the artery which is proximal to its vertebral branch. The left subclavian artery develops from the left seventh intersegmental artery alone. The two common carotid arteries, and the proximal parts of the two internal carotid arteries are developed from the third aortic arches. The rest of the internal carotid arteries develop from the two paired dorsal aortæ headwards of the third arches, and from cranial extensions of the aortæ. The external carotid arteries arise as new outgrowths from the aortic sac, after the disappearance of the first and second aortic arches, but as the heart descends their origins are transferred to the third aortic arches. During descent of the heart the origin of the innominate artery is drawn headwards towards that of the left common carotid artery, just as happens in the case of the left subclavian artery. In this way all three vessels come to arise close together near the summit of the arch of the adult aorta.

DISCUSSION

This case is of interest from two points of view—namely, the right-sided cervical aortic arch, and the vascular ring in the thorax. An almost identical case was reported in 1947 by Beaven and Fatti, showing both these features, and having an anatomical arrangement of the aorta and its branches similar to the case reported here, except that the arch of the aorta did not rise so high in the neck, reaching only to the thyroid cartilage.

The Cervical Aortic Arch.—The only other case of an aortic arch in the right side of the neck so far reported is that of Beaven and Fatti (1947), but I know of one other case not yet reported. So far as I am aware, there is no record of an aortic arch being found in the left side of the neck. The clinical importance of this condition is that it simulates an aneurysm of the right common carotid artery, and this diagnosis was made before operation in both these cases. Beaven and Fatti changed the diagnosis at the time of operation to one of aneurysm of the innominate artery, because the right recurrent nerve curved around the dilated vessel, the compression of which caused the right radial and carotid pulses to disappear. Compression exerted for 5 min. produced no signs of cerebral ischæmia. A glance at *Fig.* 639 explains these findings, for the aorta must have been compressed above the origin of the left common carotid artery. Ligation resulted in death of the patient from acute left ventricular failure after 15 min. In the present case the large pulsating vessel was, fortunately, recognized as the aorta at the time of the first operation upon the neck, and no ligature was applied.

Features which may be of help in distinguishing this condition from an aneurysm of the right common carotid artery are first the young age of the patient. My case was a boy of 5 years of age when first seen, and that of Beaven and Fatti was a girl of 9 years of age. In both the swelling had been present for several years, and in both it increased in size with the passage of time. Aneurysms are rare in childhood, and a pulsating swelling in the neck at this time of life should be suspected as being due to an abnormal aortic arch.

The second feature which may help to distinguish these conditions is the type of pulsation. In the case reported here the swelling showed two pulsations to each heart-beat, while Beaven and Fatti (1947) described the pulsation in their case as having "a peculiarly sinuous expansile" nature. These features are probably caused by asynchronous pulsation of the afferent and efferent limbs of the cervical aortic arch, and their presence should make one suspect this condition. A systolic murmur and thrill were present in this and in Beaven and Fatti's case, but they do not help in differentiating an arch of the aorta in the right side of the neck from an aneurysm of the right common carotid artery.

The third feature which should distinguish the two conditions is the effect of compression of the pulsating swelling on the peripheral arterial pulses. Compression of an aneurysm of the right common carotid artery should obliterate only the pulses in the distribution of this artery, perhaps also the pulses in the right arm if the subclavian artery is obliterated during compression of the aneurysm. The pulses in the left side of the neck, the left arm, and both legs will not be affected. Compression of a right-sided aortic arch in the neck will obliterate the pulses in the right side of the neck, the right arm, and both legs, but will not obliterate the left carotid pulse, and it may or may not obliterate the pulses in the left arm. The left common carotid artery arises from the ascending aorta in the chest in these cases, below the point at which the aorta is compressed in the neck, so that pulsation in the carotid vessels on the left side of the neck is unaffected. In both this case, and in that of Beavan and Fatti, the left subclavian artery arose from a diverticulum of the left paired dorsal aorta, as the last branch of the 'aortic arch', and compression of the aorta in the neck would have obliterated pulsation in the left arm. It is theoretically possible that the left subclavian artery might arise in common with the left common carotid artery from a left innominate artery, as a reference to *Fig.* 642 will show. In such a case pulsation in the left arm would not be eliminated by compression of the aortic arch in the neck. The safe rule is to examine the femoral pulses when the cervical swelling is compressed. If

the condition is a cervical aortic arch the femoral pulses will disappear; if an aneurysm of the right common carotid artery the femoral pulses will be unaffected. At the second operation upon the neck of my patient it was demonstrated that compression of the vessel in the neck did obliterate the femoral pulses, so clinching the diagnosis. Palpation of the pulses in the left arm, while the cervical swelling is compressed, will determine the origin of the left subclavian artery either from a left innominate artery or from an aortic diverticulum.

The fourth feature which will distinguish between an arch of the aorta in the right side of the neck and an aneurysm of the right common carotid artery is the presence or absence of symptoms and signs of a vascular ring in the thorax. Both this and the other reported case exhibited such features. My patient suffered from dyspnœa and cyanosis on exertion during childhood which disappeared as he grew up. When a young adult he developed fairly severe dysphagia. The girl of 9 years of age reported by Beavan and Fatti (1947) complained of dyspnœa on exertion and of slight dysphagia on extending the neck. In the case reported here there was a typical indentation of the posterior wall of the œsophagus. It will be remembered that the axillary pulse was weak, and that the brachial, radial, and ulnar pulses were absent on the left side when he was first seen. Subsequently pulsation appeared at the left wrist, but it was always weaker than on the right side. The findings at thoracotomy (*Fig.* 639) explained why this was so. The left subclavian artery arose from the tip of a conical-shaped aortic diverticulum, and the angle of junction of the two was acutely angled forwards by the tense ligamentum arteriosum. Moreover, with each heart-beat this angle was pulled forwards and inwards. It is reasonable to suppose that this anatomical arrangement interfered with pulsation in the left arm. It is possible that there may have been stenosis at the point of attachment of the ligamentum arteriosum, though this was not apparent on examination of the vessels from without. The lesson is, that the alteration of the pulses in the left arm depended upon a congenital abnormality associated with the presence of a vascular ring, and a similar finding in other cases would lead one to suspect this diagnosis. In 1 of the 11 cases of right-sided aortic arch with retro-œsophageal segment reported by Bedford and Parkinson (1936) the left radial pulse was scarcely palpable.

Finally, the two conditions may be differentiated by angiography. This procedure clearly demonstrated the arch of the aorta in the neck in my patient (*Fig.* 638).

The developmental error responsible for the high right-sided arch of the aorta in the neck is of interest. Beavan and Fatti (1947) suggested that the right third aortic arch, and not the fourth, persisted to take part in the formation of the arch of the aorta. If this were to happen and the right ductus caroticus were to persist, a high right-sided aortic arch would occur from which the external and internal carotid arteries would arise as separate branches—the state of affairs which was present in both their case and the one reported here. It was because the two carotid vessels arose separately from the aortic arch that they advanced the theory that the third right aortic arch had persisted. This argument is not necessarily valid because if the fourth arch and also the ductus caroticus on the right side were to persist then the external and internal carotid arteries would also arise as separate branches from a right-sided aortic arch. This may occur on the left side with a normally developed left-sided arch of the aorta. With either arrangement the right subclavian artery would form the third branch of a right-sided aortic arch, as in this case and in that of Beavan and Fatti. If the left subclavian artery arose from the eighth segment of the left paired dorsal aorta, as in both these cases, then it would form the fourth branch of the aorta. If it arose from the ventral part of the left fourth aortic arch it would arise from a left-sided innominate artery. The arrangement of the branches of the aorta cannot tell us whether the third or the fourth embryological aortic arch has taken place in its formation. Neither can the fact that the recurrent branch of the vagus nerve hooks around the arch, as it was proved to do in the case of Beavan and Fatti (1947), for it will bear this relationship whether the third or the fourth arch persists.

There are two embryological possibilities which might explain the high position of the arch of the aorta. The first is the suggestion advanced by Beavan and Fatti (1947). The second is that the elements partaking in the formation of the aorta, including the right fourth aortic arch, fail to descend. The embryonic arches surround the developing pharynx and they are primarily cervical structures; only later do they, and the heart, descend into the chest. There appears to be no way of telling which of these possibilities is the correct one. The fact that the heart itself, and the point of fusion of the two dorsal aortæ, and the ligamentum arteriosum all occupy their normal positions in the thorax is against the theory of failure of descent, which would have to affect only the elements forming the right-sided aorta. I therefore favour the view put forward by Beavan and Fatti (1947). If this is correct then the elements forming the abnormal aorta are the truncus arteriosus, the right horn of the aortic sac, the right third aortic arch, the ductus caroticus, the third to eighth segments of the right paired aorta, and the unpaired aorta. The ductus caroticus represents the first two segments of the paired aorta.

The Vascular Ring in the Thorax.—Various classifications of anomalies of the arch of the aorta and its branches capable of obstructing the trachea and œsophagus have been suggested, the most important of which are those of Gross and Ware (1946), Neuhauser (1946), Edwards (1948 a), and Kirklin and Clagett (1950). Edwards (1948 a) used the functioning double aortic arch (*Fig.* 640) as the basic pattern for these anomalies, and he explained the normal and abnormal arrangements of the aorta and its branches as being due to atresia of various portions of the aorta on one or other side. Reference to *Figs.* 640–643 will enable the reader to ascertain the portions which have disappeared in the normal and various abnormal patterns.

Edwards (1948 a, b) divided the abnormalities into two groups according to whether the ductus arteriosus is placed on the left or right side, i.e., attached to the left or right pulmonary artery, the term 'ductus arteriosus' being used whether or not

its obliteration has occurred. He pointed out that the ductus is usually, but not invariably, on the same side as the upper part of the descending aorta, and that thoracotomy should be performed on whichever side it is situated. Kirklin and Clagett (1950), adopting the concepts of Edwards, classify the abnormalities into two groups according to whether the upper part of the descending aorta is on the left or the right side. They adopt this procedure because the position of the aorta can be identified, whereas that of the ductus can only be inferred, and also because in a few instances the ductus arteriosus is placed on the side opposite to that of the upper part of the descending aorta. These authors give special attention to the latter type of case and quote Gruber (1912), Hermann (1928), Gordon (1947), Neuhauser (1949), and Halpert, Snoddy, Bohan, and Freede (1949) as reporting cases of this nature. Neuhauser (1949) reported 5 cases with a right-sided aortic arch and upper part of the descending aorta, but with a ligamentum arteriosum running behind the œsophagus to be attached to the left pulmonary artery, so completing a vascular ring which caused severe stridor and dysphagia. These patients were successfully operated upon by Gross.

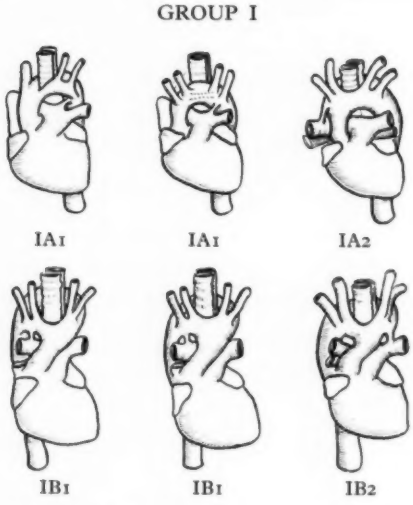


FIG. 640.—Diagram illustrating varieties of case falling into group I, characterized by functioning double aortic arch.

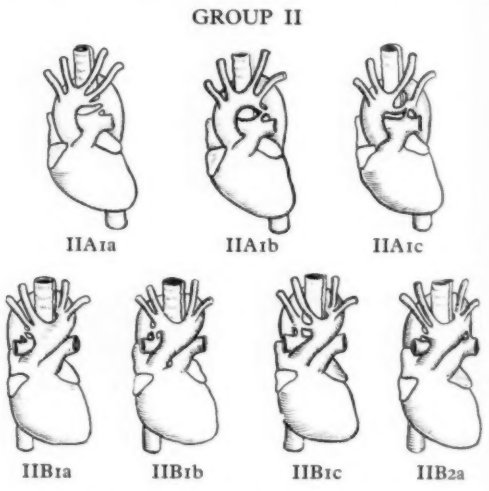


FIG. 641.—Diagram illustrating varieties of case falling into group II, characterized by double aortic arch with partial atresia of one arch.

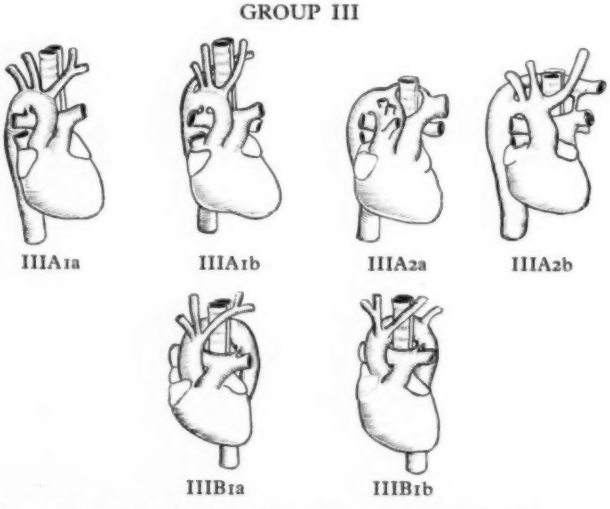


FIG. 642.—Diagram illustrating varieties of case falling into group III, characterized by right-sided aortic arch.

I have modified the classifications of Edwards (1948 a) and of Kirklin and Clagett (1950) to make the classification shown in *Table I*, which to me seems more convenient and easier to remember. All the possible variants in this classification have not yet been described, as indicated in the table. References to some of the rarer types are given. *Figs.* 640–643 illustrate many of the possible types.

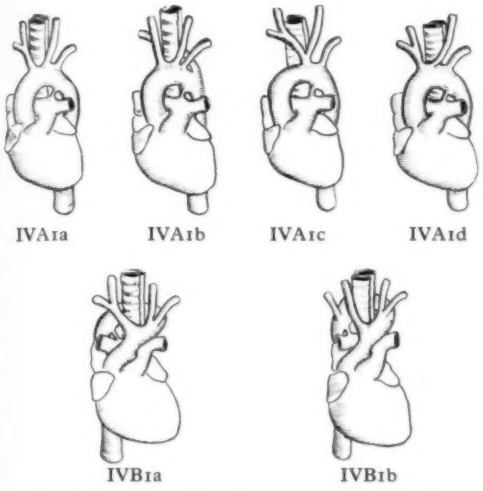


FIG. 643.—Diagram illustrating varieties of case falling into group III, characterized by left-sided aortic arch.

Four factors are used in this classification—namely: (1) The anatomy of the arch of the aorta; (2) The side occupied by the upper part of the descending aorta; (3) The side of the ligamentum arteriosum (i.e., the obliterated or open ductus arteriosus); and (4) The arrangement of the branches of the arch of the aorta, especially the subclavian artery. When the upper part of the aorta is on the right side its lower part gradually crosses to the left behind the œsophagus, usually at the level of the eighth or ninth thoracic vertebra (Bedford and Parkinson, 1936; Edwards, 1948 a), to reach the aortic hiatus in the diaphragm.

Cases are placed into groups I to IV according to whether there is a functioning double aortic arch, a double aortic arch with partial atresia of one of them, a right-sided aortic arch, or a left-sided aortic arch. Cases of functioning double aortic arch have been reported from this country by Lewis and Parry (1948), Stevenson (1950), Dolton and Jones (1952), d'Abreu, Astley, and Parkes (1952), Smithells (1953), Jones and Walker (1955), and Apley, Ross, and Walker (1957). Each group is divided into subgroups A and B by the position of the upper part of the descending aorta. When a double aortic arch is present, whether functioning or with partial atresia of one arch, if the descending aorta is on the left side the condition falls into subgroup A; if on the right side into subgroup B. If the arch of the aorta is single then the case is classified as A if the upper part of the descending aorta is on the same side as the arch, and as B if on the opposite side. Subgroups A and B are divided into 1 and 2 according

Table I.—CLASSIFICATION OF ANOMALIES OF ARCH OF AORTA AND ITS BRANCHES

I. Double aortic arch, functioning. The posterior arch is usually the larger, but the anterior may be (Gordon, 1947; d'Abreu and others, 1952), or both may be of equal size (Stevenson, 1950; Jones and Walker, 1955).
  A. With left descending aorta.
    1. With left ligamentum arteriosum (described).
    2. With right ligamentum arteriosum (described).
  B. With right upper part of descending aorta.
    1. With right ligamentum arteriosum (Gross, 1953).
    2. With left ligamentum arteriosum (Hermann, 1928).

II. Double aortic arch, with partial atresia of one arch. Atresia of the anterior, but not of the posterior, arch has been described.
  A. With left descending aorta.
    1. With left ligamentum arteriosum.
      a. Atresia between left subclavian artery and aortic insertion of ligamentum arteriosum (the usual type, Arkin, 1936).
      b. Atresia between aortic insertion of ligamentum arteriosum and junction of right and left aortic arches (rare type).
      c. Atresia between left common carotid and subclavian arteries (not yet described).
    2. With right ligamentum arteriosum.
      a.
      b. } As in group IIA1: none of these have yet been described.
      c.
  B. With right upper part of descending aorta.
    1. With right ligamentum arteriosum (not yet described).
      a. Atresia between right subclavian artery and aortic insertion of ligamentum arteriosum (not yet described).
      b. Atresia between aortic insertion of ligamentum arteriosum and junction of right and left aortic arches (not yet described).
      c. Atresia between right common carotid and subclavian arteries (not yet described).
    2. With left ligamentum arteriosum.
      a.
      b. } As in group IIB1: none of these have been described.
      c.

III. Right aortic arch.
  A. With right upper part of descending aorta.
    1. With right ligamentum arteriosum.
      a. With left subclavian artery from innominate artery (mirror image of normal pattern).
      b. With left subclavian artery from aortic diverticulum (described).
    2. With left ligamentum arteriosum.
      a. With left subclavian artery from innominate artery (Neuhauser, 1949).
      b. With left subclavian artery from aortic diverticulum (Halpert and others, 1949; Humphreys, 1948).
  B. With left descending aorta.
    1. With left ligamentum arteriosum.
      a. With left subclavian artery from innominate artery (described).
      b. With left subclavian artery from aortic diverticulum (described).
    2. With right ligamentum arteriosum.
      a. With left subclavian artery from innominate artery (not described).
      b. With left subclavian artery from aortic diverticulum (not described).

IV. Left aortic arch.
  A. With left descending aorta.
    1. With left ligamentum arteriosum.
      a. With right subclavian artery from innominate artery (normal pattern).
      b. With right subclavian artery from aortic diverticulum (common variant).
      c. With aberrant right innominate artery crossing the front of the trachea (Gross, 1953, 1955).
      d. With aberrant left common carotid artery crossing the front of the trachea (Gross, 1953, 1955).
    2. With right ligamentum arteriosum.
      a. With right subclavian artery from innominate artery (not described).
      b. With right subclavian artery from aortic diverticulum (not described).
  B. With right descending aorta.
    1. With right ligamentum arteriosum.
      a. With right subclavian artery from innominate artery (described).
      b. With right subclavian artery from aortic diverticulum (described).
    2. With left ligamentum arteriosum.
      a. With right subclavian artery from innominate artery (not described).
      b. With right subclavian artery from aortic diverticulum (not described).

to whether the ligamentum arteriosum is on the same side as the upper part of the descending aorta or on the opposite side. Finally, if the arch of the aorta is single, each of these subgroups is further divided into a and b categories according to whether the subclavian artery of the side opposite to the arch of the aorta arises from an innominate artery or from an aortic diverticulum, i.e., from the ventral end of the embryonic fourth aortic arch, or from the persistent eighth segment of the paired dorsal aorta. Thus a case designated IIIB1b would have a right-sided aortic arch with the descending aorta and ligamentum arteriosum on the left side, and the left subclavian artery arising from an aortic diverticulum. Reference to *Fig.* 639 shows that my case is one of this type, and so was that reported by Beavan and Fatti (1947). The normal arrangement of the arch of the aorta and its branches would be classified as IVA1a (*Fig.* 643) and the mirror image of this pattern as IIIA1a (*Fig.* 642).

A glance at the classification will show that under group IVA1 four subdivisions are given, namely a to d. Subgroups c and d are very slight variants of the normal arrangement, but are of importance because they may, as Gross (1953) has shown, result in compression of the trachea. In variety c the innominate artery arises too far to the left and crosses in front of the trachea to reach the right side, while in variant d the left common carotid artery arises too far to the right and must cross in front of the trachea to reach the left side. In both variants the two vessels may arise from a common stem.

Clinical Features.—These vascular abnormalities may be divided into three clinical groups, namely: (1) Those that may compress the œsophagus; (2) Those that may compress the trachea; and (3) Those that may compress both the œsophagus and trachea. Group 1 comprises aberrant subclavian arteries falling into types IVA1b (*Fig.* 643) and IIIA1b (*Fig.* 642). Although the subclavian artery nearly always passes behind the œsophagus (85 per cent), it occasionally passes between the œsophagus and trachea (15 per cent), and rarely in front of the trachea (5 per cent), as shown by Holzapfel (1899). Group 2 comprises anomalous innominate and left common carotid arteries of types IVA1c and IVA1d (*Fig.* 643). Gross (1953, 1955) reports cases of severe tracheal compression due to such abnormalities. The other abnormalities shown in the classification may all compress both the trachea and œsophagus and fall into clinical group 3.

Other cardiovascular anomalies rarely accompany those under consideration here. Jones and Walker (1955) reported a case associated with Fallot's tetralogy, and could find only 5 previous cases with associated anomalies, 3 with Fallot's tetralogy, 1 with complete transposition, and 1 with non-functioning left ventricle.

The symptoms which may result from vascular abnormalities have been described by numerous workers, including Bayford (1794), Wolman (1939), Gordon (1947), Gross (1945, 1946, 1953, 1955), d'Abreu, Astley, and Parkes (1952), Dolton and Jones (1952), Apley (1949), Brown (1950), Taussig (1947), Steen and Douglas (1955), and Apley, Ross, and Walker (1957), while Smithells (1953) discusses the differential diagnosis of congenital stridor. Full bibliographies are given by Edwards (1948 a, b) and by Kirklin and Clagett (1950). Symptoms are most likely to occur in infancy and old age. During infancy tracheal obstruction tends to dominate the clinical picture, but in old age dysphagia is the more striking feature. The patient reported here was unusual in that dysphagia developed first in young adult life. Bedford and Parkinson (1936) suggest that the development of arteriosclerosis may aggravate compression of the œsophagus after the age of 50 years.

In infants tracheal obstruction causes inspiratory and expiratory stridor, usually from birth, dyspnœa, cyanosis, intercostal and suprasternal inspiratory retraction, and crowing respiration. The infant uses its accessory muscles of respiration and tends to keep its head extended. The respiratory difficulty is aggravated by feeding, during which alarming cyanotic attacks may occur, and flexion of the head may result in complete respiratory obstruction. These infants are prone to recurrent respiratory infections, most probably attacks of inhalation pneumonitis, which may result either in death or in permanent damage to the lungs. Difficulty in swallowing may date from birth, but more often it first appears when the infant is weaned. The child may be hungry and eager to feed, but soon after starting it exhibits distress, and after several attempts to swallow it refuses to try again. Some of the food may be regurgitated. Nutrition can usually be maintained by giving frequent small feeds.

Adults may suffer severe dysphagia. In the patient reported here the disability was a considerable nuisance, but his nutrition was not impaired. The woman described so graphically by Bayford (1794), who used the term 'dysphagia lusoria', died from staravation caused by an aberrant right subclavian artery, at the age of 62 years. She had had dysphagia all her life and "fancied that she nourished within her a voracious animal, and attributed all her uneasiness to the fury with which this half-starved monster fell upon each morsel in its passage to the stomach". At last, "worn out with fatigue and famine, she sank into the grave".

The Radiological Features.—The differential diagnosis of the various vascular abnormalities is made chiefly by radiological examination. The radiological changes have been elucidated by the researches of many workers, including Assmann (1924), Evans (1936), Bedford and Parkinson (1936), Fray (1936), Arkin (1936), Garland (1938), Paul (1948), and Neuhauser (1946, 1949). They depend upon the vascular abnormality present. Straight radiographs, including penetrating films, will usually determine the anatomy of the aortic arch and descending aorta, but give little information about the branches of the aorta or the ligamentum arteriosum. It is usually possible to say whether the arch of the aorta is on the right side or left side, or if it is double. Neuhauser (1949) points out that when the arch is double the main mass of the aorta is more or less equally divided between the two sides, whereas when it is single the main mass is on one side, and the aortic knuckle will be seen either on the right or left side. When the aortic arch is double, an abnormal knuckle may be seen on both sides. When there is an aortic arch on one side,

with a retro-œsophageal segment and aortic diverticulum leading to a descending aorta on the side opposite to the arch, an appearance suggestive of two aortic knuckles may also be seen, that on one side produced by the arch of the aorta and that on the other by the aortic diverticulum (Arkin, 1936; Bedford and Parkinson, 1936). Films of suitable penetration may demonstrate narrowing of the lower third of the trachea, especially in lateral projection, while radioscopy may show under-aeration of the lungs during inspiration and over-aeration during expiration.

Valuable information is obtained by outlining the œsophagus and trachea with contrast medium. A double aortic arch constricts both the œsophagus and trachea from three aspects. The œsophagus is indented on both sides by the two arches, and from behind by the posterior arch. The latter is the right arch when the upper part of the descending aorta is on the left side, and the left arch when it is on the right side. The posterior indentation is transversely placed, usually at the level of the third or fourth thoracic vertebra, and it pulsates. The size is determined by that of the posterior arch and so affords evidence on the relative size of the two arches. When the posterior arch is large, as in the cases of double aortic arch with partial atresia of the anterior arch, described by Arkin (1936), the œsophagus and trachea are displaced grossly forward in the right anterior oblique view. In this view also the posterior arch can be seen as a rounded pulsating opacity behind the œsophagus, within which is a darker opacity, produced by an aortic diverticulum, if present. The anterior arch may also be seen as a pulsating opacity in front of the trachea in the right oblique view. Further information on the relative size of the two arches is obtained from the size of the right and left indentations on the postero-anterior film. The tracheogram shows definite indentations on either side of the lower third of the trachea, made by the two aortic arches, and in front by the pulmonary arteries. According to Gross (1953) this combination of impressions upon the trachea and œsophagus is almost certain evidence of some sort of vascular ring, provided there is no mediastinal mass. When such a ring is occasioned by an anomaly with an aortic arch on only one side the trachea and œsophagus may still be compressed from the same three aspects. The commonest anomaly of this sort is type IIIB1a or b (*Fig.* 642). The trachea and œsophagus are compressed by the arch of the aorta on the right side and the ligamentum arteriosum on the left side. The retro-œsophageal segment of the aorta compresses the œsophagus from behind and the pulmonary arteries compress the trachea from in front. The impressions on the right side of the trachea and œsophagus will be large and pulsatile, but those on the left side will be small and non-pulsatile. The posterior œsophageal indentation will be large, transverse, and pulsatile. A case of type IVB1a or b (*Fig.* 643) would show wide pulsatile indentations on the left side, made by the arch of the aorta, and smaller ones on the right side, caused by the ligamentum arteriosum. The posterior œsophageal impression would have characteristics similar to those produced by a case of type IIIB1a or b (*Fig.* 642).

Vascular rings caused by anomalies in which the ligamentum arteriosum is on the side opposite to the upper part of the descending aorta produce a different radiological picture. Neuhauser (1949) reported 5 cases in which the arched and descending portions of the aorta were on the right side, but the ligamentum arteriosum was on the left side (type IIIA2a and *Fig.* 642). The ligamentum ran behind the œsophagus from the left pulmonary artery to the right descending aorta just below the arch. The barium swallow showed an indentation on the right side made by the arch of the aorta, a shallow, barely perceptible indentation at the same level on the left side, made by the ligamentum arteriosum, and a narrow, non-pulsatile indentation posteriorly, also caused by the ligamentum arteriosum.

An aberrant right subclavian artery arising as the fourth branch of a left-sided arch of the aorta (persistent eighth segment of right fourth aortic arch), type IVA1b (*Fig.* 643), or the mirror image of this anomaly, in which the left subclavian artery arises as the fourth branch of a right-sided aortic arch, type IIIA1b (*Fig.* 642), causes an indentation of the posterior wall of the œsophagus which is obliquely placed, running from the distal part of the arch of the aorta towards the apex of the opposite side of the chest. The indentation is pulsatile, relatively small, and usually situated opposite the third or fourth thoracic vertebra. As already mentioned, an aberrant subclavian artery occasionally passes between the œsophagus and trachea, and rarely in front of the latter.

Anomalous innominate or left common carotid arteries (types IVA1c and d, *Fig.* 643) indent the front of the trachea, but the œsophagus is normal. Gross (1953, 1955) describes a long and pronounced indentation of the anterior aspect of the lower third of the trachea, with ill-defined upper and lower borders, demonstrable by injecting contrast medium. It is difficult to tell whether the indentation is produced by an anomalous innominate or left common carotid artery, for it is seen in the lateral view so that the direction of the obliquely running vessel cannot be determined. A filling defect of this nature, in the absence of a mediastinal mass, suggests a vascular abnormality.

It is usually possible to determine whether the arch of the aorta is on the right or left side, or if it is double, and also to ascertain the position of the upper part of the descending aorta. If two arches are present it is often possible to estimate their relative sizes. The position of the subclavian artery can sometimes be demonstrated and that of the ligamentum arteriosum demonstrated or inferred. With such knowledge an exact anatomical diagnosis of the vascular anomaly can often be made. If there is still doubt the diagnosis can be proved by angiography, but this is not often necessary. Stevenson (1950) has shown the usefulness of horizontal body section radiography. Now that these conditions are amenable to surgical treatment an exact pre-operative diagnosis is important because it has an influence on the operative approach which must be used.

TREATMENT

At present there is no surgical procedure available for the treatment of an aortic arch in the right side

of the neck. The replacement of the malformed arch and its branches by a graft would be a formidable undertaking. The shortness of the carotid vessels on the right side would enhance the difficulties. This condition must not be mistaken for an aneurysm of the right common carotid artery, for ligation of the ascending aorta would prove fatal.

The surgical treatment of vascular anomalies which constrict the trachea and œsophagus has been pioneered by Gross (1945, 1946, 1953, 1955). He has reported results in 26 cases of double aortic arch, 18 cases of right aortic arch with left ligamentum arteriosum, 9 cases of anomalous innominate artery, 5 cases of anomalous left common carotid artery, and 12 cases of aberrant subclavian artery, 11 on the right side and 1 on the left side. As a result of the work of Gross (1945, 1946, 1953, 1955), Edwards (1948 a, b), and Kirklin and Clagett (1950) it can be said that offending vessels, including the ligamentum arteriosum, must be divided or dislocated, and that any accompanying bands of soft tissue must also be divided. Lewis and Parry (1948) considered that surgical cure of double aortic arch might prove possible, but were obviously unaware that Gross had reported a case successfully treated in 1945.

The anomalies being discussed cause symptoms only in a minority of the patients in whom they occur (Gross, 1945; Gordon, 1947). In others symptoms are too mild to warrant surgery, while infants tend to improve as they grow. There is, however, a proportion of patients who are dangerously disabled and in whom surgery is essential in order to prevent pulmonary damage, inanition, or death.

If surgery is required to relieve tracheal obstruction in infancy, Kirklin and Clagett (1950) advise that it should be carried out as early in life as possible. This policy will both relieve obstruction of the trachea and œsophagus and allow of normal growth and development. If treatment is unduly delayed, prolonged compression of the trachea may prevent proper development of the cartilaginous rings, so that partial collapse of the trachea will persist after operation and exclude a perfect result.

Pulmonary infection and malnutrition must be corrected before operation is performed. Intratracheal anæsthesia is essential because the trachea is both narrow and soft in these cases. The operative approach depends upon the anomaly to be treated. Gross (1953) advises operating through a left anterolateral thoracotomy regardless of the anomaly present, but Edwards (1948 a, b) and Kirklin and Clagett (1950) advise opening the chest on the side of the ligamentum arteriosum. Edwards (1948 a) states "usually the structure or structures which must be treated surgically are either the ductus arteriosus or some structure which lies in the same side of the body as the ductus; therefore, it is advantageous for the surgeon to know, pre-operatively, on which side the ductus lies in order that he may perform the thoracotomy on that side". He goes on to say that the ductus arteriosus usually lies on the same side as the upper part of the descending aorta, so that the latter structure may guide the surgeon as to the proper side on which to operate. But, as pointed out by Kirklin and Clagett (1950), the ductus arteriosus is occasionally located on the side opposite to the upper part of the descending aorta. In these anomalies characterized by a vascular ring encircling the trachea and œsophagus the upper part of the descending aorta is more often on the left side than on the right side, and the ligamentum arteriosum is usually, but not invariably, on the same side as the descending aorta. In my opinion thoracotomy should be performed on the side of the ligamentum arteriosum when this can be identified, otherwise the upper part of the descending aorta should be taken as the guide. A standard posterolateral thoracotomy incision gives excellent access for most conditions, but anomalous innominate and left common carotid arteries may be approached more easily via an anterolateral incision.

The structures which require division or displacement depend upon the anomaly present. An aberrant right or left subclavian artery, of types IVA1b (*Fig.* 643) or IIIA1b (*Fig.* 642), arising as the fourth branch of an aortic arch on the left or right side, and crossing behind the œsophagus, should be divided. Division of the first part of the subclavian artery is usually safe, though on rare occasions the blood-supply to the arm is jeopardized. Anomalous innominate or left common carotid arteries (types IVA1c and d) should be dislocated forwards by silk sutures passing through their adventitia and through the sternum. They must not be divided. Care is taken that the sutures do not pass deeper than the adventitia.

When a vascular ring is present it must be broken. In all cases the ligamentum arteriosum should be divided, and in certain cases other vessels will require division in addition. In the case of a functioning double aortic arch the smaller arch is divided. Most commonly a smaller left anterior arch is combined with a descending aorta and ligamentum arteriosum on the left side (type IA1). This arch should be divided between the left common carotid and left subclavian arteries, and not proximal to the former. If after doing this the anterior part of the arch and the left common carotid artery form a tight band which compresses the trachea, they should be hitched forwards by sutures, as described for anomalous innominate or left common carotid arteries. If the posterior arch is the smaller then it should be divided behind the œsophagus. In this type of case the anomaly may be completely overlooked as the large anterior arch may appear to be normal and it obscures the smaller posterior one from view (Gordon, 1947; d'Abreu and others, 1952). Blincoe, Lowence, and Venable (1936) found this arrangement in only 2 out of 40 cases. Amongst 41 cases in which the size of the two arches could be determined, Jones and Walker (1955) found the right arch to be the larger in 26, the left arch to be the larger in 10, and the two arches to be of equal size in 5, including their own case. This series includes that of Griswald and Young (1949). When there is a double aortic arch with partial atresia of one arch (type II, *Fig.* 641), the atresic portion is divided, together with the ligamentum arteriosum. In cases of types III (*Fig.* 642) and IV (*Fig.* 643), in which the arch of the aorta on one side is connected to the descending aorta on the other side by a retro-œsophageal segment, division of the ligamentum arteriosum alone will break the ring and relieve constriction. The same is true of

those cases in which the arched and descending portions of the aorta are on one side, but the ligamentum arteriosum is attached to the pulmonary artery of the opposite side. In this event the ligamentum arteriosum completes a vascular ring by passing behind the œsophagus (*Fig.* 642) and its division will relieve symptoms. This was the state of affairs in the 5 cases of type IIIA2a reported by Neuhauser (1949), and successfully operated upon by Gross.

Results of Surgical Treatment.—Complete symptomatic relief following surgery is the rule. Five of the 70 infants and children operated upon by Gross (1955) died. These were all in the group, 26 in number, with double aortic arches, and their ages ranged from 1 month to 3 years. Two deaths were due to operative hæmorrhage. In one of these patients the anterior arch was divided proximal to the left common carotid artery, where it is large and short. Division and ligation should always be performed distal to this artery. A third death was due to cerebral œdema the day after operation in a seriously ill child who had been in an oxygen tent for four months prior to admission to hospital. Two died of pneumonia three and ten days after operation. Gross (1955), speaking of double aortic arch, states that "the overall results have been exceedingly dramatic and prove beyond doubt that an operative attack on this vascular abnormality has great merit". He goes on to say, "it is generally highly desirable to undertake surgical correction of the vascular anomaly whenever it is discovered".

SUMMARY

The case is reported of a young man whose aortic arch occupied the right side of his neck, simulating an aneurysm of the right common carotid artery, and who had a vascular ring in the chest, causing dysphagia, which was relieved by surgery.

An account of the development of the aorta and its branches is given, and the occurrence of the arch of the aorta in the right side of the neck is considered.

The clinical features of an aortic arch in this site, and its differential diagnosis from an aneurysm of the right common carotid artery, are described.

A classification of vascular anomalies which may compress the trachea and œsophagus is suggested, their clinical and radiological features are detailed, and their surgical treatment is briefly discussed.

Acknowledgements.—My grateful thanks are due to Professor Lambert Rogers, who asked me to see this patient; and to Professor Lambert Rogers and Professor Cecil Lewis, who have allowed me to use their records.

My thanks are also due to Mr. R. Marshall, who photographed the illustrations, and to Miss June Williams, who drew *Fig.* 639 and *Figs.* 640–643.

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THE LYMPHATICS OF THE BREAST*

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THE importance of the lymphatic system in the spread of carcinoma must not be overemphasized, but the present treatment of breast carcinoma in its early stages, both surgical and radiotherapeutic, is based upon the anatomy of the regional lymphatics and an accurate knowledge of these pathways is therefore necessary.

The accepted anatomical descriptions of the lymphatics of the breast are largely the result of post-mortem injection studies; for technical reasons, however, these methods are unsatisfactory because trunk lymphatics of the breast cannot be exposed for direct injection and indirect filling from interstitial injection is very irregular; retrograde injection of normal lymphatics is virtually impossible.

The only certain method of demonstrating lymphatics of the breast, and the only possible method of assessing the distribution of lymph drainage, is by intravital techniques; the observation of dyes and markers as they are transported by the natural flow of lymph from an injection site in the breast to the regional nodes. The spread of carcinoma is of limited value as a method for the study of lymphatic drainage and can only be used for this purpose at an early stage of the disease.

In the present work the anatomy of the lymphatics of the breast has been reviewed using intravital techniques in 88 patients.

HISTORY

Gaspard Aselius (1627) was the first to demonstrate the lacteals, which he found in a recently fed dog. The similarity between the chyle and milk stimulated seventeenth-century observers to seek a direct communication between the lacteals and the lymphatics of the breast in order to explain lactation. Pecquet (1654) saw milk-laden lymphatics from the breasts of a lactating bitch enter the thorax; but he mistook the direction of lymphatic flow in these and replied to a contemporary, Riolan: "Audiat Sane . . . let him learn what path there is for chyle, unmixed with blood, to the breasts . . . Let him learn also that there is in the living body, just as in the sky, a milky way . . .".

The lymphatics of the human breast were first described in detail by Cruikshank (1786): ". . . two

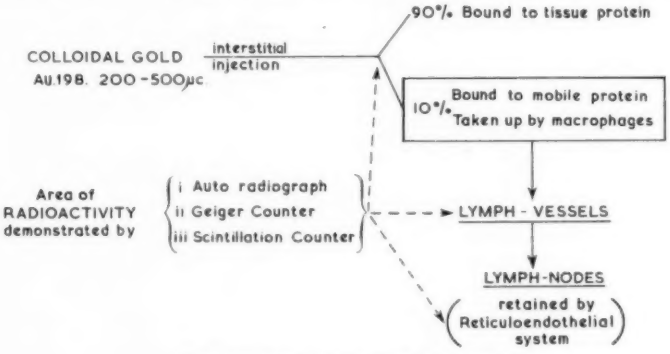


FIG. 644.—The radio-active colloidal gold method.

sets of absorbents, one accompanying the external thoracic artery and vein and the other the internal thoracic (mammary) vein. The external absorbents arise from the nipple and from the external part of the mamma, from the integuments and the tubuli lactiferi. They run outwards towards the axilla and sometimes pass through small glands halfway between the nipple and the axilla. . . . Some of the external thoracic vessels pass over pectoralis major and go to the glands under the middle of the clavicle. . . . The internal mammary absorbents arise from the posterior part of the mammæ and perforate in many places the intercostal muscles. . . ."

Mascagni (1787) published many excellent illustrations of his preparations; these do not show the lymphatics of the breast, but he commented: "From the superficial tissue of the thorax and epigastrium, besides the lymphatics which run to the axillary glands, other vessels of the same kind accompany the epigastric, intercostal, and internal mammary vessels."

A hundred years later Sappey (1885) described the lymphatics of the breast. He believed, as a result of a mercury injection of a lactating breast, that the parenchyma drained into the 'subareolar plexus'. He specifically denied that any lymph-vessels left the posterior surface of the breast or that any lymph-vessels from the breast reached the internal mammary chain.

Heidenhain (1889), Stiles (1892), and Sampson Handley (1906) studied the spread of cancer of the

* Part of an essay for which the 1957 Moynihan Prize of the Association of Surgeons was awarded and of a Hunterian Lecture delivered at the Royal College of Surgeons on May 27, 1957.

breast and formulated the theory of tumour dissemination by "lymphatics of the deep fascial plexus".

Poirier, Cuneo, and Delamere (1903) recognized three accessory channels in addition to the main pathway, described by Sappey, from the subareolar plexus to the axillary glands. They were: (1) Vessels leaving the parenchyma of the breast and passing directly to the axilla—these they considered inconstant. (2) The interpectoral pathway of Grossman (1896) and Rotter (1899). (3) The internal mammary pathway of Cruikshank (1786) and Mascagni (1787). Poirier, Cuneo, and Delamere add: "It is important to remark that we should not exaggerate the importance of the internal mammary channel. Though its presence is anatomically indisputable, facts none the less show that in the early stage of cancer of the breast invasion of the retrosternal glands is exceptional."

Mornard (1916) classified lymphatics of the breast into six somewhat ill defined groups. Gray (1938) used post-mortem injections of thorotrast. He denied the existence of the deep fascial plexus.

In 1947 R. S. Handley and Thackray published the first of a series of papers showing the incidence of invasion of the internal mammary lymph-nodes by carcinoma of the breast at an early stage of the disease. This was investigated by biopsy, or excision, of the internal mammary chain during radical mastectomy. By 1954 these authors had examined 150 cases; one-half of the tumours in the medial half and one-fifth of the tumours in the lateral half of the breast had metastasized to the internal mammary lymph-nodes and in 10 per cent of each group the axillary nodes were free from invasion.

Morl (1952) used ante-mortem intramammary injections of indian ink. He showed that all quadrants of the breast drained into the sternal lymph-nodes. He was unable to assess contralateral drainage because he used bilateral injections.

Hultborn, Larsen, and Raghnult (1955) injected radioactive colloidal gold into the breast several days before radical mastectomy and measured the lymph-node uptake post-operatively, both in the specimen and the patient. He concluded that the axillary nodes received nearly all the breast lymph; the parasternal uptake he estimated as 0–3 per cent. Many of his patients had received pre-operative radiotherapy.

METHODS USED IN PRESENT STUDY

The lymphatics of the breast have been demonstrated by intravital methods, injecting dyes and markers into the breast tissue; these are transported to the regional nodes by the natural flow of lymph. The details of these methods have been discussed elsewhere (Turner-Warwick, 1959).

A single injection is made into the breast tissue, usually some distance from the nipple, so that the path of the local lymphatics can be studied in relation to the subareolar plexus. If more than one injection site is used, contralateral drainage and quadrant distribution cannot be assessed.

The volume of the injected fluid is 1–2 ml. and may contain more than one of the substances discussed below; a local anæsthetic is always included to make the injection pain-free.

The lymph-flow from the resting breast is very slow and the intramammary injection should be done at least one hour before the time of observation in order to allow time for lymphatic uptake.

The drainage pathways can be assessed over periods of up to five days or more by using radio-active colloidal gold. If the lymphatic drainage of the normal breast is to be assessed it is important that the lymph drainage should be uncomplicated by

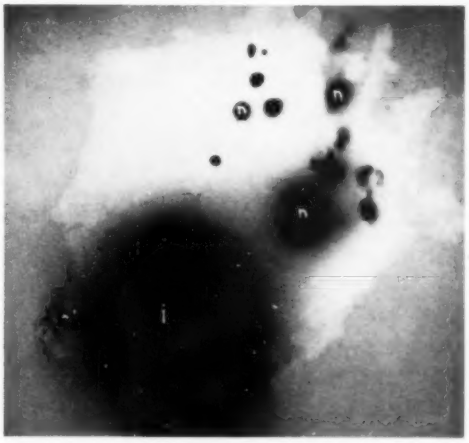


FIG. 645.—Autoradiograph of a mastectomy specimen. Three hundred microcuries of radio-active colloidal gold Au^{198} were injected into the lower outer quadrant (i) six hours before mastectomy. Fourteen nodes (n) are demonstrated as radio-active areas. A soft-tissue X-ray shadow has been superimposed.

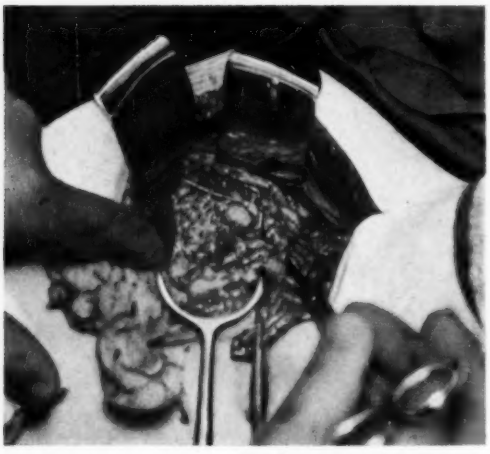
the presence of a tumour or other local pathology in the breast or the regional lymph-nodes.

During an operation the lymphatics within the operation field can best be demonstrated by a diffusible dye such as patent blue (*Fig.* 646); 1 ml. of a 3 per cent solution (rendered isotonic) is usually sufficient to show breast lymphatics and reduces the transient post-absorption body blueness to a minimum. Patent blue tends to diffuse out of the lymphatics during mass clearing of a large specimen with alcohol and benzine; more permanent dyes, such as pontamine sky blue, survive this process, but their persistence *in vivo* renders them undesirable for clinical use.

A method has been devised for the demonstration of regional lymphatic trunks in a cleared specimen as a result of previous intravital absorption. Iron dextran or saccharate injected into the tissues is partially taken up by lymphatics and can be precipitated in them as prussian blue by fixation of the specimen in acid-potassium-ferrocyanide (Turner-Warwick, 1955, 1959). Dionosil, a particulate diodone, has been used to demonstrate the extent of the local injections radiographically; it has no special tendency to enter lymphatics, but has none of the disadvantages of thorotrast in clinical use. Thorotrast is a useful medium for the post-mortem demonstration of lymphatics, but during intravital absorption it becomes too diluted with lymph to provide radiographic contrast in the vessels.

Radio-active colloidal gold provides the most accurate method available for studying the proportional distribution of the lymphatic drainage

(*Fig.* 644). Small doses are absorbed almost exclusively by lymphatics, largely retained by the lymph-nodes, and easily measured. The colloidal Au^{198} precipitates upon tissue proteins on injection. Although 90 per cent of the dose remains at the site of injection,



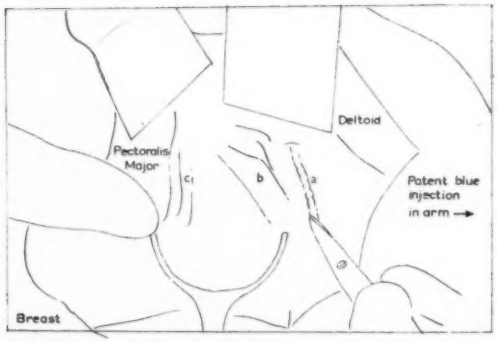


FIG. 646.—An arm-draining lymphatic (**a**) in axilla (in line with scissors) demonstrated during axillary dissection by a pre-operative patent blue injection into the arm. This particular lymphatic lies above the axillary vein (**b**) and would be unlikely to carry breast lymph under normal conditions. (**c**) Position of main lateral thoracic lymphatic pathway from breast.

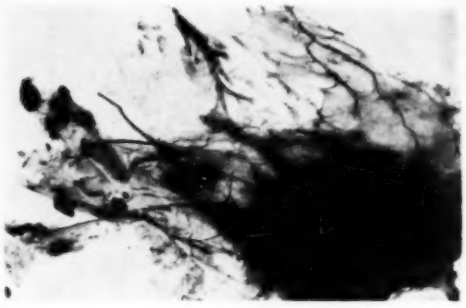
about 10 per cent is fixed to mobile proteins which are absorbed by lymphatics because their molecular weight exceeds 20,000; much of this mobile protein complex is retained by the reticulo-endothelial tissue of the lymphatic pathway. Au^{198} has a half life of 2·7 days and an injection of 100–300 μc. in its colloidal form provides good demonstration of the regional drainage by autoradiograph and quantitative estimation by counting methods.

An autoradiograph of a specimen (*Fig.* 645) is obtained by placing the partially fixed specimen in a polythene bag, sandwiched between two X-ray films for a few hours. The radio-active areas are registered on the plates by self-exposure and a soft-tissue radiograph is superimposed before removal of the film, in order to orientate the active areas in the specimen.

THE ARRANGEMENT OF THE LYMPHATICS

It has been shown that there is nothing peculiar about the arrangement of the lymphatics of the breast for, as in other parts of the body, they tend to accompany the blood-supply.

The breast receives its blood-supply from three main sources, the majority coming from the axillary vessels and the internal mammary vessels with a



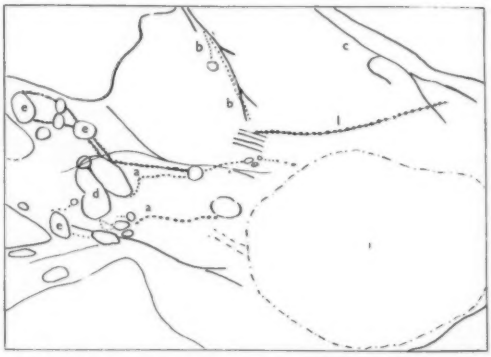


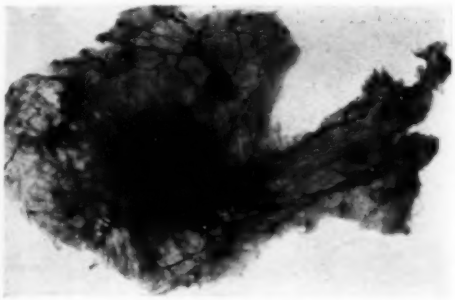
FIG. 647.—Cleared mastectomy specimen showing lymphatic pathways demonstrated by a pre-mastectomy injection (iron/prussian blue method). **i**, Injection site; **a**, Main axillary pathway; **b**, Interpectoral pathway (pectoralis major dissected away); **c**, Branches from internal mammary vessels; **l**, Lymphatic vessel passing to fourth intercostal space anteriorly; **d**, Large lymph-node replaced by growth and therefore showing no uptake compared with uninvaded nodes (**e**).

minor contribution from the lateral perforating branches of the intercostal vessels.

The lymphatic drainage in these three directions is approximately proportional to the blood-supply; most of the lymph from the breast drains to the axillary nodes, a considerable proportion to the parasternal nodes. The posterior intercostal nodes have not previously been shown to drain breast lymph; under normal circumstances, however, they only inconstantly receive a very small proportion.

The lymphatics of the main axillary pathway arise in the lobules of the breast and most of them drain directly to the lateral thoracic group of lymph-nodes (Rouvière's external mammary group) which

extend from the axillary trail *of the breast* to the apex of the axilla; the so-called 'subareolar plexus' plays no essential part in this pathway. The course of these lymphatics is not easy to illustrate. They run within the substance of the breast rather than on its superficial or deep surface, and as they run laterally they are joined by tributaries and pass through the



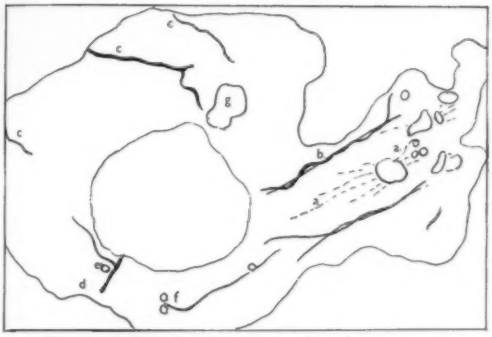


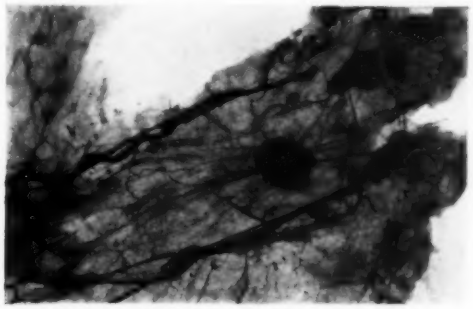
FIG. 648.—Cleared mastectomy specimen (pre-mastectomy injection; iron/prussian blue method) showing: **a**, Main axillary lymphatic pathway; **b**, Branches of the lateral thoracic blood-vessels; **c**, Mammary branches of the internal mammary vessels; **e**, Lateral perforating vessels from the fifth intercostal space, a lymphatic accompanied these and a paramammary lymph-node (**d**) lay on its course; **f**, Another paramammary lymph-node in the fourth intercostal space; **g**, Carcinoma.

axillary fascia within the thickness of the axillary tail. Once in the axilla they tend to lie on the medial wall, but at no time do they run in the deep fascial plane.

The main breast lymphatics run approximately parallel to the lateral thoracic vessels, but independently of them, especially proximally, where the main efferent lymph-vessels of the lateral thoracic group pass upwards under the lower border of the axillary vein between it and the first rib. Even in obese patients there is relatively little fat around these vessels and they may be overlooked during an axillary clearance unless particular attention is paid to them. Most of the axillary lymph-flow from the breast passes through the lateral thoracic group, but even in the normal axilla, however, some frequently reaches the subscapular group.

The main lymph-vessels draining the breast, therefore, run on the inferomedial and anterior aspects of the axillary vein and are common to the arm drainage. It is unusual for brachial lymph-vessels above the vein (*Fig.* 646) to be stained by an intramammary injection unless the axillary pathways are partially blocked as a result of malignant deposit or irradiation.

The Intercostal Pathway.—The internal mammary lymph-nodes lie along the internal mammary vessels, extending upwards from the fifth intercostal



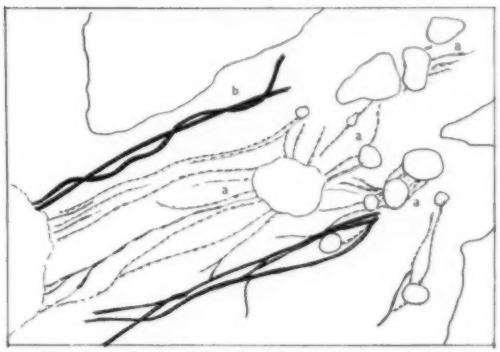


FIG. 649.—Details of the axillary lymphatics from the same specimen as *Fig.* 648.

space to the retroclavicular glands. They usually lie between the costal cartilages, but not infrequently behind them (*Fig.* 650). These lymph-nodes receive lymph not only from the lymphatics accompanying the anterior perforating vessels, but also from those accompanying the lateral perforating branches of the upper intercostal vessels.

The upper three or four intercostal spaces have large internal mammary lymph-nodes anteriorly and relatively small posterior intercostal lymph-nodes. The posterior intercostal lymph-nodes lie between the necks of the ribs, close to the vertebræ. Below the fifth space the internal mammary chain is poorly represented. There is a tendency to a "watershed" in the lymphatic drainage of the intercostal spaces, the line which becomes progressively more anterior in the lower spaces; thus lateral perforating lymphatics which accompany the intercostobrachial nerve and lateral perforating blood-vessels in the upper three or four spaces tend to drain anteriorly into the internal mammary chain while those entering lower spaces may drain into the posterior intercostal nodes.

The lateral perforating lymphatics from the breast are usually proportional to the size of the

blood-vessels which they accompany, but lymphatics accompany the intercostobrachial nerves irrespective of blood-vessel size (*Figs.* 648, 651, 652). Perforating lymphatics in the upper intercostal spaces constitute at least a theoretical lymphatic communication between the axillary and internal mammary pathways.

FIG. 650.—Breast-draining lymph-nodes of the right internal mammary chain demonstrated in a cleared anterior chest-wall specimen by the prussian blue method from an intravital iron injection into the right breast. A large lymph-node lies behind the second costal cartilage in this specimen and two more behind the fourth.

The perforating lymph-vessels leave the posterior surface of the breast and pass through the fascial plane without plexiform arrangement in the retro-mammary tissue planes. They are best demonstrated during a mastectomy if the vascular bundles are ligated close to the chest wall; the lymphatics become distended with coloured lymph if the pre-operative intramammary injection site of diffusible dye is gently massaged (*Fig.* 651). These lymphatic vessels are very difficult to demonstrate by passive injections.

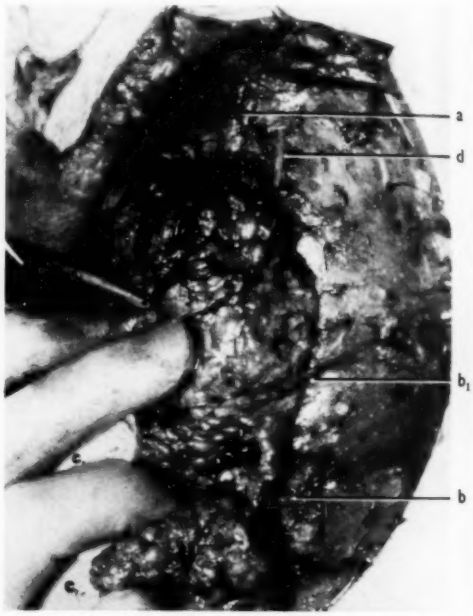


FIG. 651.—Main axillary (**a**) and lateral perforating lymphatics (**b** and $\mathbf{b_1}$) demonstrated during a mastectomy by pre-operative intramammary injection (**c**) containing patent blue. (**d**) intercosto-brachial nerve. (The lymphatics were blue in colour and the beaded lymphatic ($\mathbf{b_1}$) can be seen.)

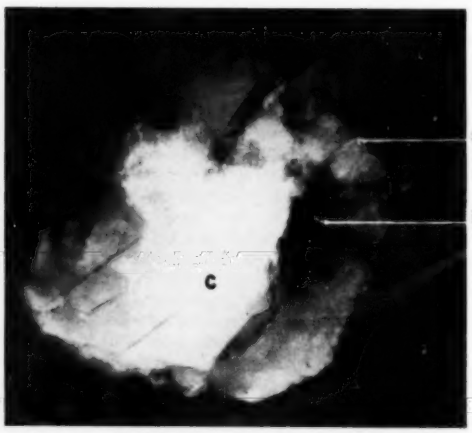


FIG. 652.—Radiograph of the specimen seen in *Fig.* 651 showing the same lymphatics outlined by thorotrast after massage of the injection site (Gray's (1938) method).

The Interpectoral Pathway.—Most of the lymphatics which leave the posterior surface of the breast pass through the pectoralis major to reach the internal mammary chain and axilla. The lymph-vessels accompanying the acromio-thoracic vessels pass through the interpectoral lymph-nodes on their way to the upper axillary chain (*Figs.* 647, 653). This pathway cannot always be demonstrated.

No lymphatics of importance traverse the pectoralis minor muscle (*Fig.* 654), but there are lymph-nodes in front of it (interpectoral), above it (subclavicular), and behind it (lateral thoracic).

The Subareolar Plexus.—The subareolar plexus was originally described by Sappey (1885) in a lactating breast, and many authors have subsequently concluded that most of the lymph from the breast drains centripetally to this area and thence to the axilla.

The present study suggests that the importance of the lymphatics of the subareolar region may have

been overemphasized. The subcutaneous network in this area communicates with the lymphatics around the lactiferous ducts and although they are, therefore, naturally included in a mastectomy excision for carcinoma, they do not represent an essential confluent point in the pathway of the lymphatic drainage of resting breast tissue (*Fig.* 656). The duct lymphatics are important as a secretion-absorbing mechanism, especially during pregnancy.

As recently as (1953) Grant, Tabah, and Adair reported a case in which there was a subareolar concentration of pontamine sky blue following several premastectomy injections around the periphery of the breast; this was thought to be due to the subareolar lymphatic plexus, but there is an alternative explanation for this unusual appearance. A peripheral intramammary injection normally stains the overlying skin, but not the more distant nipple area; isolated nipple staining is occasionally seen, however, and in this series it has been due to dye

FIG. 653.—Breast-draining lymphatics (**a**) accompanying branches of the acromiothoracic blood-vessels (**b**), within pectoralis major. (The beaded lymphatics were demonstrated by an intramammary injection and the iron prussian blue method; their blue contents clearly distinguished them from the blood-vessels in the specimen.)

entering the lactiferous system (*Fig.* 655). About 10 per cent of random injections into the breast fill the milk ducts in this way.

The Deep Fascial Plexus.—There is no evidence to support the suggestion that minute lymphatics on the fascial plane represent a normal lymphatic pathway from the breast to the regional glands. This theory, widely held at one time and still repeated in some current descriptions, must be abandoned.

Passive injections may succeed in demonstrating some extremely fine lymphatics on the fascial plane which are better regarded as the lymphatic drainage of the fascia. On no occasion have they been demonstrated by intramammary injections which readily show the important pathways from the breast. A few quite large lymphatics leave the posterior surface of the breast, accompanying the larger perforating blood-vessels. These pass across the retromammary space, entering the pectoralis major or intercostal spaces, with no suggestion of a plexiform arrangement. These perforating vessels are much larger than the fine fascial lymphatics and usually pass between them without communication.

Fascial spread is unusual early in the dissemination of a tumour; in the later stages it is probably mechanical. The study of the spread of advanced carcinoma is misleading material upon which to base ideas of the anatomy of

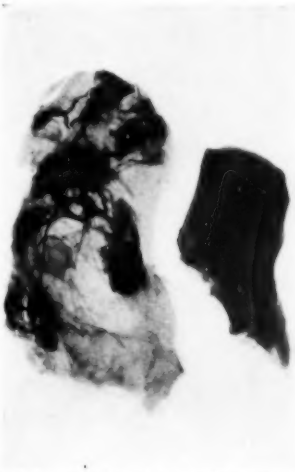


FIG. 654.—Lymphatics of the axilla (iron prussian blue method). Cleared specimen; the pectoralis minor has been dissected from the surrounding axillary tissue. No lymphatics of importance traverse it.

lymphatics or the pathways of early spread of the tumour.

The minute lymphatics which drain the fascial plane are, therefore, of no importance in the pathways of lymphatic drainage of the breast and play no part in the early spread of breast cancer, but the penetrating lymphatics which pass vertically between them are of considerable importance.

The Subcutaneous Lymphatics.—The main trunks of the subcutaneous lymphatics form a coarse mesh network over the breast and do not differ from those elsewhere in the body. They lie in the same plane as the superficial venous plexus of the breast which is well demonstrated during pregnancy; some are inevitably lifted during mastectomy even with a thin skin-flap. They extend in the same plane across the midline, over the clavicles, and down onto the abdominal wall; they anastomose with the deeper lymphatics of the breast, especially in the nipple region. This system of lymphatics drains only a small proportion of lymph from the breast, but is of considerable importance in the spread of carcinoma.

After an intramammary injection of radio-active colloidal gold there is a considerable spread in the subcutaneous tissue beyond the area of simple diffusion; this takes place mechanically along breast-tissue planes, and by lymphatics. *Fig.* 656 illustrates the value of keeping skin-flaps thin and excising a good margin of skin, but even a wide skin excision to

the limit of practicability cannot wholly eradicate this pathway.

Lymphatics from the breast very rarely enter the para-xiphisternal area: this postulated pathway of spread to the abdominal cavity is of no importance

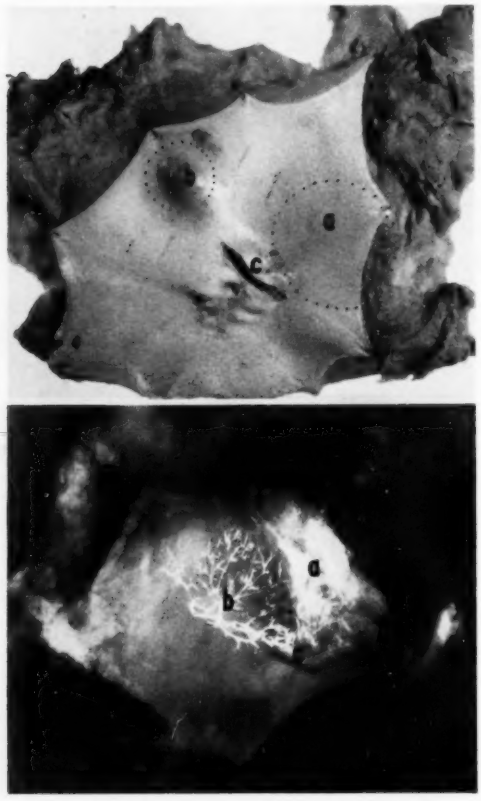


FIG. 655.—Mastectomy specimen and radiograph. **c**, Carcinoma. **a**, Skin staining over the site of peripheral injection of Evans blue and thorotrast. **b**, Distant staining beneath nipple (unusual). The radiograph of the specimen shows that the dye reached the nipple by the lactiferous system.

in the spread of carcinoma of the breast, and the paraxiphisternal lymph-nodes do not usually pick up radio-active gold injected into the breast (*Fig.* 659) unless the main pathways are obstructed. In a late spread the deposits in the lower internal mammary chain may effect retrograde communication with the peritoneal lymphatics just as the intercostal system on a whole has access to the pleural cavity.

The Retroclavicular Lymph-nodes.—The lymph-nodes of the so-called supraclavicular group receive lymph from the breast by upward extension along the axillary and internal mammary pathways; the direct pathway is insignificant. The proximal course of the main pathway is, in fact, retroclavicular, lying behind the medial third of the clavicle rather than above it. This is important if local irradiation is planned.

Most of the lymph from the arm drains along lymphatics on the anterior and medial aspects of the axillary vein, and these are interrupted by removal of the breast-draining lymphatics. A few lymph-vessels from the arm lie well above the axillary vein and

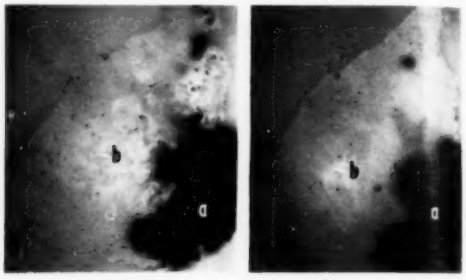


FIG. 656—**A**, Autoradiograph of an ellipse of skin removed from a radical mastectomy specimen. **a**, Diffusion staining over the site of Au^{198} injection. **b**, Pin marking nipple. The skin has been dissected off the specimen with a thin layer of subcutaneous tissue nowhere exceeding ¼ in. in thickness. There is no radio-activity in the subareolar area, but scattered areas elsewhere. **B**, Autoradiograph of the same ellipse of skin after removal of the subcutaneous tissue (comparable with a thin mastectomy skin-flap) showing the resulting reduction of the outlying radio-active areas.

these are not usually concerned with breast drainage (*Fig.* 646).

Contralateral Drainage.—There is no significant drainage of lymph from the breast to the contralateral axilla or the contralateral internal mammary chain under normal conditions (*Fig.* 657). The arrangement of the lymphatic drainage of the breasts of bitches is apparently different; Berg and Christophersen (1956) found that the contralateral parasternal pick-up in these animals sometimes exceeded the ipsilateral.

Clinically, contralateral deposits of growth are not infrequently seen in late cases, but are unlikely to occur until the ipsilateral drainage is impaired by lymphatic ablation by growth, surgery, or irradiation.

The Distribution of Lymph-flow.—Both the axilla and the internal mammary chain receive lymph from all quadrants of the breast; there is no striking tendency for any particular quadrant to drain in one direction. The lateral perforating vessels are most frequently seen after injection in the lower outer quadrant, and these may account for some of the internal mammary chain.

The ipsilateral axillary nodes commonly receive more than 75 per cent of the total lymph from the breast. The remainder drains into the ipsilateral internal mammary chain.

Radio-active gold studies in 12 selected patients with no local pathology in the breasts or lymph-nodes indicate that there is a wide individual variation in the drainage to the internal mammary nodes (*Figs.* 658, 659) and in the actual uptake in the different interspaces (*Fig.* 660). In one of the twelve drainage studies in this series the internal mammary uptake from a lower outer quadrant injection exceeded that in the axilla (*Fig.* 660), while another showed no internal mammary uptake at all after ten hours' drainage of an injection in the same quadrant.

The ipsilateral internal mammary chain undoubtedly represents an important pathway of lymph drainage from both the lateral and the medial halves of the breast. The posterior intercostal nodes of the third and fourth spaces received lymph from the breast in one of the 12 patients, but the amount was less than 2 per cent of the total drainage. The great majority of patients have no drainage at all from the breast in this direction unless the main pathways become obstructed.

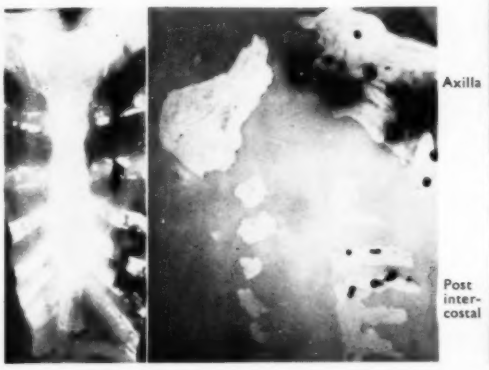


FIG. 657.—Autoradiograph of sternum and axillary and posterior intercostal tissue. Ipsilateral lymph-nodes in all three areas show radio-active uptake as a result of six hours' drainage of an intramammary injection containing colloidal Au^{198} into the lower outer quadrant; there is no contralateral drainage. The posterior intercostal lymph-nodes only occasionally drain breast lymph.

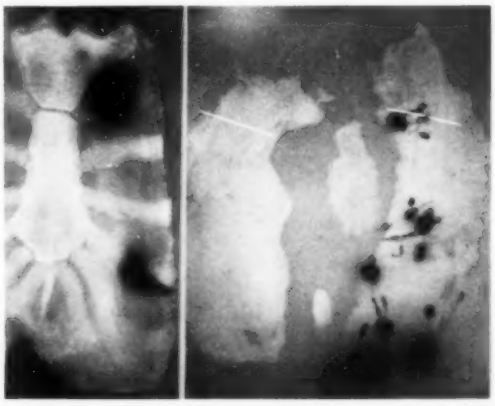


FIG. 658.—Autoradiograph of sternum and axillary contents showing greater pick-up in the axillary nodes after 24-hour drainage of an intramammary injection of 600 μc. of colloidal Au^{198} into the lower outer quadrant. Some lymphatic vessels are clearly demonstrated in the axilla. The pin marks the level of the clavicles. (There was no uptake in the posterior intercostal nodes of either this case or that shown in *Fig.* 659.)

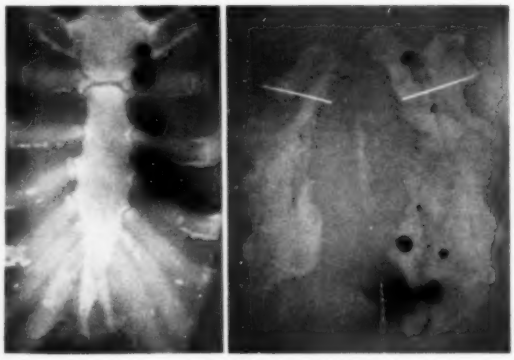


FIG. 659.—Autoradiograph of sternum and axillary tissue. In this case the internal mammary lymph-node uptake from a lower outer quadrant intramammary injection exceeded that in the axilla (24 hr. 600 μc. colloidal Au^{198}). (The patterns of breast lymph drainage shown in *Figs.* 657–659 show variations of the normal pathway. There was no tumour or other pathology in the breasts or lymph-nodes of these cases.)

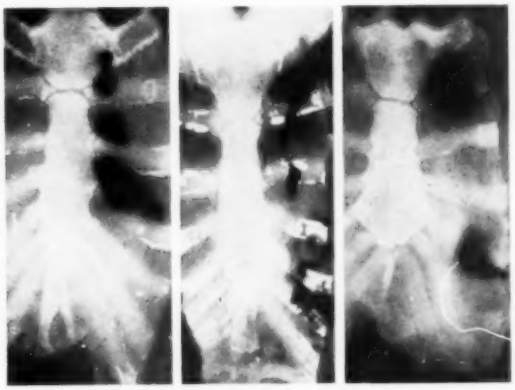


FIG. 660.—Autoradiograph of the three sternal specimens showing the normal variation in the distribution of the breast-draining lymph-nodes of the internal mammary chain. (Injections of colloidal Au^{198} into the lower outer quadrant of the breast in each case.) No uptake in para-xiphisternal nodes.

SUMMARY

1. The trunk lymphatics of the breast tend to accompany the blood-supply and the proportional distribution of the breast lymph is discussed.

2. The internal mammary chain is an important pathway of the lymph drainage from both the lateral and the medial halves of the breast.

3. There is no significant drainage of lymph from the breast to contralateral nodes under normal circumstances.

4. The posterior intercostal lymph-nodes have been shown to receive lymph from the breast in a small proportion of patients.

5. The so-called subareolar plexus has not been found to play an important role in the pathway of lymph drainage of the resting breast parenchyma.

6. The minute lymphatics of the deep fascia play no part in the main pathway of the lymphatic drainage of the breast nor in the early spread of carcinoma.

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INTRALOBAR SEQUESTRATION OF LUNG, ASSOCIATED WITH FOREGUT DIVERTICULUM (ŒSOPHAGOBRONCHIAL FISTULA) AND AN ABERRANT ARTERY

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DAVIDSON (1956) was the first to demonstrate preoperatively the rare but interesting malformation-complex of accessory lung, foregut diverticulum, and an aberrant artery in a case of hiatus hernia. There have been reports of similar cases since then, and also in the earlier literature, where the anomaly was discovered at necropsy or operation. The association of sequestration of the lung with foregut diverticulum is rare, and the present case demonstrates features which may throw some light on the genesis of this complex group of malformations.

CASE REPORT

A male schoolteacher, 31 years of age, was first seen in June, 1957, in the Thoracic Surgical Unit. His main complaint was severe cough, productive of greenish sputum; this dated from an attack of whooping-cough in infancy, but early in 1957 the daily sputum output had increased suddenly. An X-ray film of the chest at the Chest Clinic had shown multiple cystic areas in the left lower lobe, consistent with a diagnosis of bronchiectasis (*Fig.* 661). He was also complaining of slight exertional dyspnœa.

Physical examination revealed marked clubbing of the fingers. The trachea was central, and there were numerous coarse râles all over the left hemithorax, while the breath-sounds were diminished over the left lower lobe posteriorly.

Bronchography was carried out: unfortunately, filling was not satisfactory owing to technical difficulties. The bronchograms were reported on as "fairly marked saccular bronchiectasis of basal bronchi, left lower lobe, with partial collapse". Further review of the bronchograms, after the correct diagnosis was made at thoracotomy, showed the basal bronchi pushed outward by the cystic area which itself was never filled; the anterior basal bronchus occupied its normal position, while the lateral basal bronchus divided into two branches and occupied the zone of the posterior basal bronchus, in the lateral projection (*Fig.* 662). Both these bronchi showed distal sacculations. The posterior basal bronchus itself was represented by a small stump in line with the main stem bronchus of the left lower lobe.

From the clinical history and the radiological findings a diagnosis of bronchiectasis of the basal segments of the left lower lobe was made.

AT OPERATION (A. W. F.): At left thoracotomy the left lower lobe was very adherent to the diaphragm, and was gradually freed by dissection, and ligation of numerous bleeding points. An abnormal vein was found running from the inferior pole of the lower lobe into the inferior hemiazygos vein which was lying against the thoracic aorta. On dividing this between ligatures, a hollow muscular-walled structure, 1 cm. across and about 10 cm. long, was encountered coming through the diaphragm, and running into the posterior basal segment. At this stage an œsophageal tube was passed by the anæsthetist to demonstrate the course of the œsophagus. It then became apparent that the tubular structure, which itself markedly resembled an œsophagus, was in itself a diverticulum, arising from the infradiaphragmatic part of

the œsophagus. The diverticulum was transected above the cupola; it contained milk clot. The distal segment was closed and returned to the abdomen. A basal segmental resection was then carried out. There was no evidence of any diaphragmatic defect. A normal-sized basal pulmonary artery was divided between ligatures. The chest was closed with drainage; the post-operative period was uneventful. His cough and sputum almost disappeared.

FIG. 661.—Postero-anterior film of the chest (heavily penetrated), showing the cystic area in the left lower lobe, behind the cardiac shadow.

FIG. 662.—Left lateral view of the bronchogram.

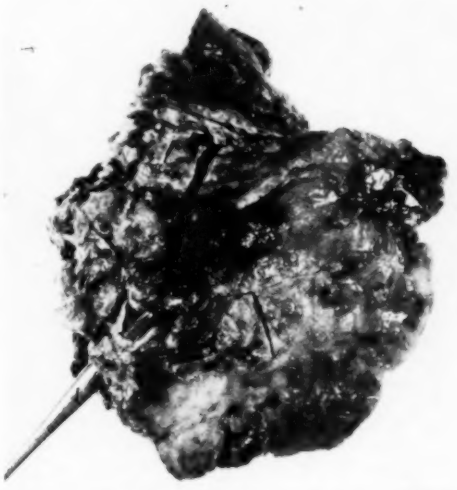

FIG. 663.—Resected specimen with hæmostat in the diverticulum. The junctional area between diverticulum and a bronchial cyst has been opened into over the point of the hæmostat. (× ½.)

FIG. 664.—Specimen bisected to demonstrate the bronchial cysts. (× ½.)

PATHOLOGY.—The resected specimen measured 9 cm. by 7 cm. at the base; it felt rubbery hard except on its lateral aspect, where it had the texture of collapsed lung. A diverticulum was running into the postero-inferior angle of the lobe (*Fig.* 663). An artery 3 mm. across entered the lung behind the diverticulum; this vessel almost immediately divided into two branches, which embraced the diverticulum, soon to be lost in an arborizing vascular network. The diverticulum was smooth on its outside; its mucosa was intact. It could be peeled off the lung parenchyma for a very short distance only. Section of the specimen showed the lobe to be made up

of numerous cysts with thick walls (*Fig.* 664); apparently normal lung tissue was encountered on the parietal aspect. The diverticulum could be traced into one of the large cysts, but was soon lost in the maze of cystic spaces. There was no evidence of ulceration or inflammation in the junctional area where the diverticulum joined the cyst.

On dissection of the bronchial tree two large bronchi were demonstrable, the anterior basal and the lateral basal, both supplying the normal lung parenchyma. The posterior basal bronchus was missing. The cysts in the lobe communicated with the stem bronchus through small perforations. Similarly, the major pulmonary arterial branches were seen to accompany the two basal bronchi while the sequestrated area was supplied by several minute arterial branches.

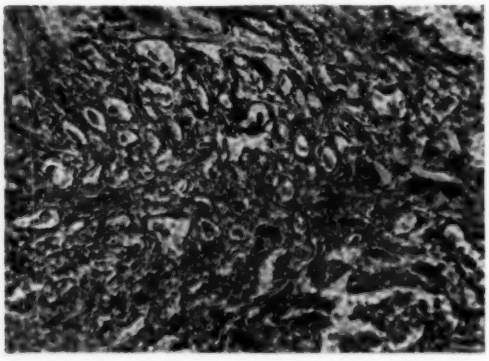

FIG. 665.—An area of interstitial fibrosis, showing cuboidal-cell metaplasia of the alveolar walls. H. and E. (× 75.)

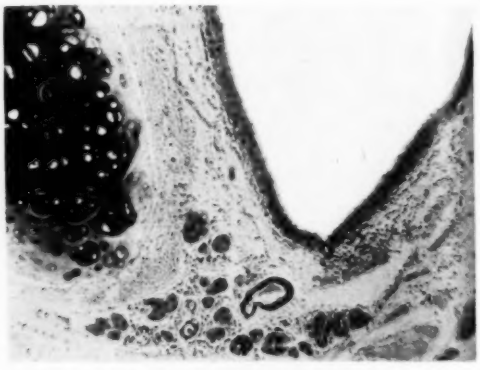

FIG. 666.—Part of the wall of the diverticulum, showing cartilage, mucous glands, and ciliated columnar epithelium. H. and E. (× 50.)

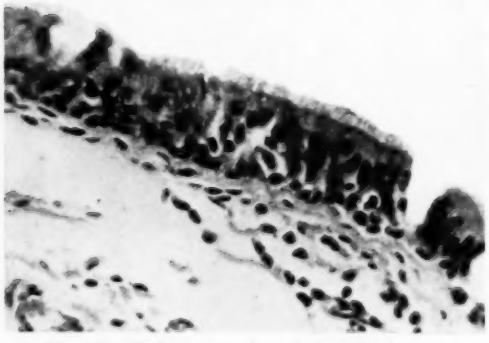

FIG. 667.—Higher-power view of the ciliated pseudo-stratified columnar epithelium lining the wall of the diverticulum. H. and E. (× 285.)

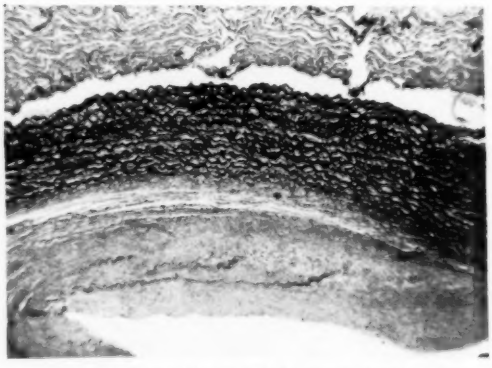

FIG. 668.—Part of the wall of the aberrant artery, showing intimal fibrosis, and abundant elastic fibres distributed throughout the media. Verhoeff-van Gieson. (× 50.)

HISTOLOGY.—Sections of the resected left lower lobe show numerous thick-walled cavities, lined by columnar ciliated epithelium; the intervening lung parenchyma shows widespread collapse, with well-marked interstitial fibrosis, and hypertrophic bands of smooth muscle (the so-called 'muscular cirrhosis' of lung). There are many areas of bronchiolar proliferation and of cuboidal-cell (reversionary) metaplasia of alveolar walls (*Fig.* 665).

Sections of the diverticulum reveal the characteristic features of a bronchus: stratified columnar ciliated epithelium, with mucus-secreting 'goblet' cells, mucous glands of bronchial type, and plates of cartilage in the wall of the diverticulum (*Figs.* 666, 667).

The aberrant artery accompanying the diverticulum has the 'elastic' structure characteristic of the aorta and its large primary branches. The thick tunica media shows a dense network of elastic fibres throughout its whole thickness, and there is no definite condensation into internal or external elastic laminæ. The lumen is narrowed by a plaque of intimal fibrosis (*Fig.* 668).

The arteries accompanying the two normal lower lobe bronchi show the usual appearance of pulmonary arteries; the media is less thick than in the aberrant artery, and there is no evidence of intimal thickening.

DISCUSSION

The respiratory tree is formed from the laryngo-tracheal groove in the floor of the foregut in the 3-mm. embryo. The groove deepens to form a tube, the lower end of which bifurcates into the two primary bronchial buds. These, by branching, eventually give rise to all the subdivisions of the respiratory tract, including bronchioles and air sacs. The right-sided bud gives off two lateral branches (upper and middle lobe bronchi), and the stem bronchus

becomes the lower lobe bronchus. On the left side only one lateral bud is present. Within the lower lobe itself the posterior basal bronchus is the final representative of the stem bronchus, while the other basal bronchi come off earlier as offshoots. Lateral budding commences in the 7-mm. embryo, and the forerunners of all the segmental bronchi are obvious at the 14-mm. stage. By this time the definitive pulmonary artery has descended into the hilum on each side.

There are just over one hundred cases of sequestration of lung in the literature, the majority of these on the left side, and mostly confined to the posterior basal segment, but a sequestrated lung with a foregut connexion is extremely rare. *Table I* summarizes the cases recorded in the literature where an œsophagobronchial or gastrobronchial fistula was associated with pulmonary sequestration. The cases fall into two categories: (1) Those with a foregut diverticulum and intralobar sequestration; and (2) Those with a foregut diverticulum and extralobar sequestration (accessory lobe).

Accessory lobe has been attributed to supernumerary budding of the developing bronchus (Eppinger, 1904), but the origin of intralobar sequestration has been the subject of controversy. Rektorzik (1861) appears to have been the first to suggest that part of the developing lung might become sequestrated or 'fractionated' during embryonic life. This fractionation theory has been elaborated by Pryce (1946), who described 7 cases of intra-lobar sequestration, associated with an aberrant artery arising from the aorta. He believed that the aberrant artery was the primary anomaly, and that the sequestration resulted from the separation of a budding bronchial tip from the embryonic bronchial tree, due to traction by the adventitious artery. He stressed the 'elastic' nature of the artery and its liability to atheromatous change, but regarded it as a combined pulmonary and bronchial artery, which he called a 'systemic pulmonary'.

Baar and d'Abreu (1949), describing 2 cases of accessory lobe, considered that the accessory lobes arose as supernumerary buds, or duplications, of the foregut, and that any aberrant arterial supply was of secondary importance. They pointed out that the vascular system plays a relatively small role in early embryonic life, and that the development or regression of vessels at this period depends on the rate of growth of the tissues they supply. Basing their view on the recent work of experimental embryologists' they further explain that accessory buds of the primitive foregut become displaced and undergo subsequent differentiation under the influence of local organizer centres. We would prefer to accept this view, and also agree with them that the structure of the aberrant artery is determined not so much by its embryological history as by considerations of hæmodynamics. In the present case the structure of the media and the atheromatous intimal change indicate that this artery is a first-order branch of the aorta, subject to the usual mechanical stresses of such an artery. No valid deductions as to its developmental history can, in our view, be made at this stage.

Whatever the nature of the primary developmental error, it is clear that the growing tip of the bronchial bud may, at some stage in its development, become dissociated from the bronchial tree. If this takes place at the 5-mm. stage the whole lung may be sequestrated, as in the case of Klebs (1874). At the 10–14-mm. stage sequestration will involve only that part of the lung derived from the advancing tip of the stem bronchus—that is, the posterior basal segment of the lower lobe. This is the more familiar type of intralobar sequestration.

Table I.—RECORDED CASES

AUTHOR	BRONCHOPULMONARY MASS	DIVERTICULUM	ABERRANT VESSELS	ASSOCIATED ANOMALIES
A. Intralobar Sequestration with Foregut Diverticulum				
1. Klebs (1874)	Total sequestration of right lung	Bronchial diverticulum, blind at œsophageal end	+	–
2. Beneke (1905)	Total sequestration of both lungs	Tracheal bifurcation and both main bronchi arising from lower œsophagus	+	–
3. Duprez, Wittek, and Dumont (1956)	Sequestration of lateral segment of right middle lobe	Œsophageal diverticulum	–	–
B. Accessory Lobe with Foregut Diverticulum				
1. Scheidegger (1936)	Left lower accessory lobe	Gastrobronchial fistula	+	–
2. Gans and Potts (1951)	Left upper accessory lobe—retropleural in position	Œsophagobronchial fistula at level of main carina	– (Arterial supply from pulmonary artery)	–
3. Davidson (1956)	Left lower accessory lobe	Œsophagobronchial fistula	+ From abdominal aorta	Hiatus hernia*
4. St. Raymond, Hardy, and Robbins (1956)	Left lower accessory lobe	Œsophagobronchial fistula	+ From abdominal aorta; venous drainage into portal system	Hiatus hernia*

* The cases with hiatus hernia had aberrant arteries arising from the abdominal aorta, which may explain the failure in the complete separation of the pleural from the peritoneal cavity.

A more difficult problem is raised by the presence, in our case, of an œsophageal diverticulum forming an œsophagobronchial fistula in the sequestered segment. The whole œsophagus is lined by ciliated columnar epithelium in early embryonic life; and the normal bronchial tree arises as an outgrowth from the foregut. Baar and d'Abreu's (1949) case of a cyst in the œsophageal wall lined by ciliated epithelium must be considered as a variety of duplication of the foregut. The diverticulum we describe may also be regarded as a duplication of the foregut. To account for its communication with the cystic pulmonary mass there appear to be two possibilities. Firstly, it may have 'captured' the terminal bud (destined to form the posterior basal segment) of the developing bronchus, which has then lost its normal attachment and acquired a new one. Secondly, the diverticulum may have acquired the potentialities, as well as the structure, of a developing bronchus, and itself differentiated into a mass of pulmonary tissue. In this case one would have to suppose that the differentiation of the normal posterior basal segment from the terminal stem bronchus had been competitively inhibited by the abnormal 'bronchus'.

SUMMARY

A case of intralobar sequestration of the lung, associated with an œsophageal diverticulum (œsophagobronchial fistula) and an aberrant 'elastic' artery, is described. It is considered that the malformation is probably a variety of duplication of the foregut.

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MULTIPLE CONGENITAL DIVERTICULA OF THE JEJUNUM

REPORT OF A CASE

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SINCE the original reports of Astley Cooper in 1807 and Meckel in 1817 numerous reports have been made of multiple diverticula of the jejunum protruding from the mesenteric aspect of the bowel into the leaves of the mesentery. These diverticula arise where the arteries penetrate the muscularis of the bowel, and are generally held to be acquired diverticula protruding through these weak points in the wall (Edwards, 1934, 1936, 1939; Case, 1920; Spriggs and Marxer, 1925, 1926; Rothschild, 1925; Fraser, 1933; and Butler, 1933). Most of the recorded cases with such diverticula have been in the middle or later decades of life when the condition has been diagnosed or discovered, and very few cases have occurred in the first two decades of life. Even less frequent are the reports of diverticula arising from the antimesenteric border of the jejunum; in all, some 10 cases have appeared in the literature, each report describing a solitary diverticulum in this site. The reports studied were those of Hanseman (1896), Helvestine (1923), Edwards (1936), Orr and Russell (1951), Major (1839), and Miller (1931). The age incidence in these reports ranged from 2 to 46 years. Two reports, those of Edwards, and Orr and Russell, describe the presence of an 'aberrant' artery coursing over the outside of the bowel from the mesentery, to divide at the very apex of the diverticulum. The diverticula in which the microscopical anatomy has been described have had a complete investment of muscle coats in 2 cases (Miller, 1931; Orr and Russell, 1951) and absent muscularis in another (Edwards, 1936). The purpose of this paper is to describe and discuss a number of such diverticula found in a child dying on the second day after delivery.

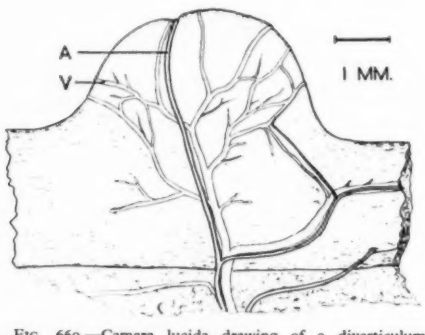

FIG. 669.—Camera lucida drawing of a diverticulum cleared in oil of wintergreen, to demonstrate the artery passing to the apex. A, The artery; V, Veins.

CASE REPORT

A male infant weighing only 4 lb. (1·75 kg.) was born at term on April 2, 1957. The mother had previously been delivered of a hydrocephalic infant in 1945 and of normal healthy infants in 1954 and 1955. At birth the

child was observed to have a cleft palate, left talipes equinovarus, a short wide neck, and broad hands with a fixed flexion deformity of the fingers. The heart-sounds were normal, but as the child exhibited repeated cyanotic attacks a congenital heart lesion was suspected. Death occurred during a cyanotic attack 42 hours after delivery. There was no history of any illness during the pregnancy.

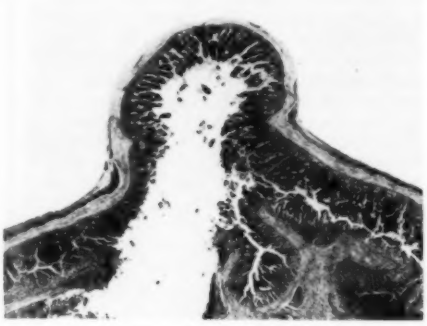

FIG. 670.—Photomicrograph of a diverticulum with irregular muscle-bundles in the wall. Phloxine-tartrazine. (× 10.)

The only abnormality was the development of hydramnios of moderate degree in the last trimester.

Necropsy Report.—P57/151. The body was that of a male baby, with intense cyanotic lividity. It was obviously small for its maturity. The neck was short and broad, but the number of cervical vertebræ was found to be normal and there was no fusion. A left talipes equinovarus was present, with a mild degree of talipes on the right. An unusual tense flexion of the fingers was present, which was not due to rigor mortis.

The heart was enlarged in relation to the size of the body, weighing 15 g. The aorta and pulmonary artery arose in transposed position from the right ventricle, the pulmonary artery overriding a very large interventricular septal defect formed by failure of upgrowth of the dividing septum. A non-valvular interatrial septal defect 0·5 cm. in diameter was also present. There was no pulmonary stenosis, the great veins opened normally, and the ductus arteriosus (0·2 cm. diameter) was still patent.

A large bony and soft-tissue defect, 1 cm. across, was present in the roof of the mouth, extending backwards to a completely divided uvula, but not involving the alveolar margin. Eight diverticula, from 0·2 to 0·5 cm. in diameter, were present on the antimesenteric border of the upper jejunum, beginning 5 cm. from the duodenojejunal junction and extending for the next 15 cm. The bowel here was 0·6 cm. in diameter. A white hardish nodule 0·3 cm. diameter was present on the antimesenteric border of the jejunum 30 cm. below the lowest of the diverticula. A Meckel's diverticulum (1 cm. long by 1 cm. in diameter) was also present 30 cm. above the ileocæcal valve, and approximately 100 cm. below the white nodule. The Meckel's diverticulum bore on one side, half-way to the apex, a small projection 0·2 cm. in diameter, around the base of which blood-vessels formed a complete anastomosing circle. The remainder of the alimentary tract appeared normal.

Hæmorrhage was present in both lateral ventricles of the brain, in the third and fourth ventricles, and in the subarachnoid space. It appeared to have arisen in the right cerebral hemisphere. This hæmorrhage was considered to be the principal cause of death, although the congenital heart lesion was also probably incompatible with continued life. No other major abnormality was found in any system on macroscopical examination.

Microscopy of the Diverticula.—Several diverticula have been cleared in oil of wintergreen and the vascular supply examined with the dissecting microscope. An artery, resembling those previously described (Edwards, 1936; Orr and Russell, 1951), has been found passing to the apex of the diverticulum and there dividing (*Fig.* 669). Sections show the presence of small irregular

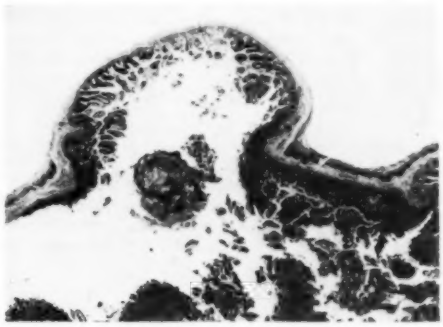

FIG. 671.—Photomicrograph of a diverticulum with no muscularis in its wall. Phloxine-tartrazine. (× 10.)

FIG. 672.—The diverticulum of the Meckel's diverticulum, cut off at the base and sectioned longitudinally. H. and E. (× 15.)

bundles of muscle in the walls of some diverticula (*Fig.* 670), with a thin collar of muscle encircling the basal portion of the diverticulum. One diverticulum is completely devoid of muscle bundles (*Fig.* 671). The wall is mainly composed of collagenous connective tissue in all the four diverticula sectioned. The muscularis mucosæ is intact and the diverticula are lined by villous small-intestinal mucosa.

The white nodule seen 30 cm. below the lowest of the diverticula is ectopic pancreatic tissue, which is enclosed between the fibres of the longitudinal muscle coat. The Meckel's diverticulum is lined by small intestinal mucosa, and has complete muscular coats, although these are thin. The projection seen on one lateral aspect is itself a diverticulum of the Meckel's diverticulum, again provided with all the muscle coats and lined by small-intestinal epithelium (*Fig.* 672).

DISCUSSION

Since this infant was only 42 hours old at death there can be no doubt that the diverticula found were congenital; moreover, their association with an abnormal artery supports their status as developmental abnormalities. It is to be noted that other well-known developmental abnormalities such as

cleft palate, talipes, and congenital heart lesions were present in association. Since these diverticula are congenital the absence or paucity of muscularis in their walls is noteworthy. The argument is often put forward that acquired diverticula produced by traction or pulsion, and coming through a weak point in the bowel wall, are devoid of muscularis; while those that are congenital and developmental, being derived from embryonic cell groups whose function is to form bowel, should be invested by all the layers of normal bowel. Edwards (1934, 1939) and Orr and Russell (1951) incline to this view, although Moynihan (1927) was not fully able to equate acquired and developmental diverticula with those that were anatomically 'false' and 'true'. Although this seems to be widely believed it is hard to see why aberrant cells should not produce tissue which is abnormal in anatomy and deficient in some structures. However this may be, this case would appear to demonstrate clearly that the rule is not a hard-and-fast one.

A number of antimesenteric diverticula of supposedly developmental origin have been associated with ectopic pancreatic tissue which is to be found at the apex of the diverticulum. Examples in the jejunum are those of Benson, Dixon, and Waugh (1943), Nauwerck, cited by Evans (1929), and possibly Major (1839). Such diverticula have variously been postulated to arise by traction on the part of the ectopic pancreas, or as part of an aberrant anlage forming pancreas and intestine from a group of totipotential cells. The embryological fact appears to be that numerous clumps of such cells can be found along the bowel in this region in early fœtal life (Moynihan, 1927; McMurrich and Tisdall, 1928); two of these form the pancreas, the remainder all disappear completely in normal development. In this case they would appear to have persisted in 9 instances, 8 forming diverticula, in which no ectopic pancreatic tissue could be found, and 1 forming an ectopic pancreatic nodule. The case thus provides further evidence in favour of a theory of origin of these diverticula from embryonic cell-rests capable of forming bowel or pancreas.

The diverticulum found projecting from the Meckel's diverticulum in this case is also presumably congenital and developmental. Such a diverticulum appears to be a very rare occurrence, the only report so far traced being that of Wilson (1955).

SUMMARY

1. The case is described of an infant with multiple antimesenteric diverticula of the jejunum associated with other congenital abnormalities. The pathological findings are described.

2. The developmental status and possible mode of origin of such diverticula is discussed, together with their association with ectopic pancreas.

3. A diverticulum of a Meckel's diverticulum present in the same case is described.

I would like to thank Dr. J. D. Roscoe for permission to publish details of her case and for help with the clinical history; and Dr. A. M. Barrett for help and encouragement in the preparation of this paper.

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MULTIPLE LYMPHATIC CYSTS OF THE MESENTERY

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Cysts of the mesentery are relatively uncommon. Beahrs and Judd (1947) record 174 mesenteric and omental cysts of various types from 1,000,000 patients at the Mayo Clinic. Thompson and Chambers (1946) estimate their incidence as 1 in 100,000. While most of the reports concern single cases, Gross (1953) was able to review the findings in 13 cases, and Handelsman and Ravitch (1954) record 4 further cases. Both these series were of mesenteric cysts occurring in infancy and childhood. They are, however, recorded in the higher age-groups and in the fœtus (Miller, 1935).

There have been various suggested classifications. Peterson (1940) differentiates cysts arising from infective processes, hydatids, or malignant disease of the mesentery from cysts of embryonic origin—serous, chylous, or sanguineous cysts or dermoids—arising from embryonic remnants, cysts of sequestrated gastro-intestinal duplications, or those arising from urogenital organs. Handelsman and Ravitch (1954)

stress the importance of separating 'chylous' cysts from those arising from enterogenous and retroperitoneal elements. Similarly, Higgins and Lloyd (1924) distinguish true mesenteric cysts as those occurring in or near the mesentery, which are not malignant or parasitic, and which do not arise in any normally placed retroperitoneal organ.

The majority of true mesenteric cysts are simple unilocular or multilocular lymphatic cysts which may contain serous or chylous fluid. There is also a rarer lymphangiomatous lesion of the mesentery, best described as a lymphangiectasia. One example occurred in Gross's series—a 7-month-old infant. MacNab and Menzies (1949) record a further case in a boy aged 18 years who presented with acute intestinal obstruction. The extent of the lesion in such cases makes it doubtful whether complete excision is possible.

Clinically, cysts of the mesentery present in several ways: as a painless enlargement of the abdomen; as an acute abdominal emergency due to volvulus, rupture, or intestinal obstruction; or as transient episodes of abdominal pain and vomiting. These episodes may occur infrequently and be of quite short duration.

Two cases of multiple lymphatic cysts of the mesentery (or lymphangiectasia) have been observed recently which are of interest in their clinical presentation and in their support for a congenital origin of the process.

CASE REPORTS

Case 1.—R. M., aged 5 years. The patient was admitted to Southampton Children's Hospital complaining of centrally situated paroxysmal abdominal pains. The attacks had occurred almost daily for four weeks. They lasted for up to half an hour. For two days before admission they had been much more severe and tenesmus was present. On occasion he vomited green fluid during the attacks, often with relief of pain. Between the attacks he had been quite free of symptoms.

On Examination.—He was doubled up with pain, which rapidly subsided. The tongue was coated. On palpation the abdomen was quite soft and gave a sensation of fullness to the right of the umbilicus, where there was slight tenderness. Bowel-sounds were normal. There were no other abnormal physical signs. The temperature was normal. No abnormalities were found on rectal examination.

Progress.—A transient attack of colic woke him during the night. He then remained symptom-free for several days, and he was discharged. Radiographs of the abdomen, examination of the stools and urine, and blood examination showed no abnormalities. Five days after discharge the pains recurred; they were similar in character and tended to wake him at night. Again he occasionally vomited. Between attacks he was quite symptom-free. On re-admission examination of the abdomen showed 'fullness' to the right of the umbilicus, thought now to be a soft, cystic, mobile tumour. Exploratory laparotomy was undertaken.

At Operation (T. R.).—A transverse incision was used. The peritoneum contained a little serous fluid. Several cysts were present in the mesentery with many surrounding smaller cystic nodules. They affected the mesentery 2 ft. from the duodenojejunal junction. The cysts were resected with 7·5 cm. of small intestine. Continuity was restored by end-to-end anastomosis. The appendix was removed, and the abdomen was closed without drainage.

Post-operative Course.—Bowel-sounds were noted on the day after operation. Recovery was uneventful. He has remained well, having now been followed for eight months since operation.

Pathological Reports.—Macroscopically the specimen consisted of a length of small intestine, 7·5 cm. in length, and a wedge-shaped portion of mesentery. The mesentery contained a cluster of small cysts, many of which contained milky fluid. Three larger cysts were present, approximately 5, 6, and 7 cm. in diameter. These were unilocular and contained clear fluid. The cystic area abutted closely on the intestine, and had been excised with a border of apparently normal mesentery.

Microscopically the mesentery contained large numbers of cysts of varying sizes (*Fig.* 673). They were lined by a flattened single-cell layer of endothelium. The cysts were most numerous towards the root of the mesentery. Some of the cysts contained fat-laden histiocytes, a small number showed red cells, while an occasional space contained both red cells and histiocytes (*Fig.* 674). Yet other cysts showed a relatively acellular coagulum. The gut adjacent to the cystic areas showed thinning of the deeper muscular layers. The general architecture of the gut layers was preserved.

The stromal tissues of the mesentery contained numerous lymphatic channels as well as small cysts, so that a continual gradation of cyst size was seen. The loose vascular areolar tissue showed some condensation about the larger cysts. Isolated groups of smooth-muscle fibres were noted in the walls of a few of the cysts (*Fig.* 675).

The stromal cellularity was increased nearer the root of the mesentery, and this area contained lymphoid aggregates. There was also a diffuse infiltration by lymphocytes and histiocytic mononuclear cells. Fibroblastic reaction was more prominent (*Fig.* 676).

Case 2.—E. C. C., aged 21 years. The patient was admitted to Southampton General Hospital complaining of abdominal pain. This was a central colic, and had occurred daily for five days. On the day of admission the pain had been severe and continuous; he vomited once. The bowels had been quite regular. In the past history there had been two episodes of paroxysmal central abdominal pain, each terminating after a few days.

On Examination.—His temperature was 101·6° F. and the pulse-rate 120. The abdomen was tender and rigid, most markedly in the right iliac fossa. Bowel-sounds were present. An old para-umbilical abdominal scar was noted. No other abnormalities were found on physical examination. Rectal examination was normal. It was decided to explore the abdomen.

At Operation (S. L.).—The abdomen was opened by a right Rutherford Morison incision. Several loops of small intestine were adherent to the anterior wall peritoneum to the right of the umbilicus. A moderate amount of seropurulent fluid was present. The terminal 2 ft. of ileum were normal. For several feet above this point the mesentery was affected by a diffuse vascular cystic neoplasia which extended to the root of the mesentery. The ileum was severely congested. Resection of the affected ileum and mesentery was carried out with high ligation of the vessels, followed by end-to-end anastomosis. The abdomen was closed with drainage.

Post-operative Course.—He was treated by intravenous fluids and gastric suction, which was continued because of paralytic ileus. Daily electrolyte levels showed hypokalæmia, the levels of potassium varying from 2·2 to 4 mEq. per litre. The ileus gradually recovered, but he also developed partial collapse of the right lower lobe necessitating bronchoscopy. The sputum yielded a heavy growth of *Staph. pyogenes* (phage type 80). He gradually improved and has remained well three months after operation.

It was learned from his father on the day following operation that the previous laparotomy at another hospital was carried out when aged 5 years for an attack of abdominal pain. The notes of this operation describe the mesentery of the small gut as containing many small,

FIG. 673.—*Case* 1. Microscopical appearances of mesentery, showing multiple cysts of varying sizes. Many are small and give the appearance of honeycombing. The cysts are lined by a simple flattened endothelium. H. and E. (× 51.)

FIG. 674.—*Case* 1. Group of cysts showing contents of fat-laden macrophages or red cells, in some cysts intermingled. H. and E. (× 168.)

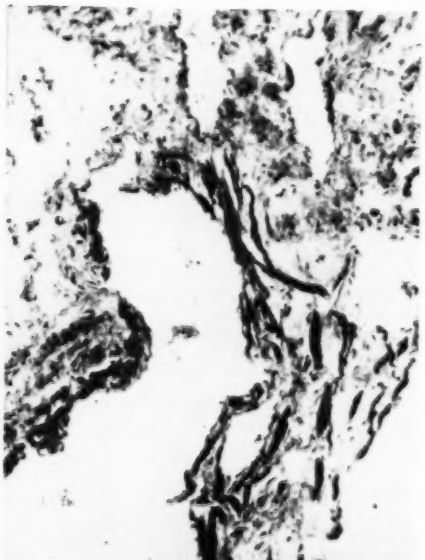

FIG. 675.—*Case* 1. Cystic spaces showing irregular strands of smooth muscle in surrounding stromal tissues. Masson's trichrome. (× 105.)

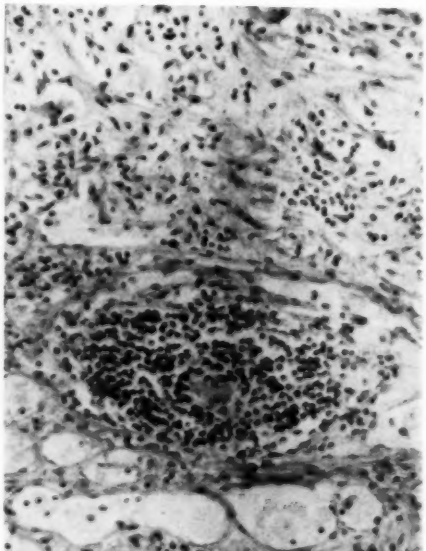

FIG. 676.—*Case* 1. Microscopical appearances of stromal tissues, showing focal and diffuse lymphocyte reaction, with fibroblast activity in other areas. H. and E. (× 282.)

smooth lymphatic cysts encroaching on the lower ileum. Appendicectomy was carried out, but no attempt was made to remove the cysts. Recovery was uneventful, and a short course of irradiation was given to the right iliac fossa. He was seen at intervals for five years and remained well.

PATHOLOGICAL REPORT.—Macroscopically the specimen consisted of a 45-cm.-length of ileum (after fixation) with attached mesentery. At four separate points in the gut wall a soft whitish tumour-like mass projected into the lumen. Each was several centimetres in diameter. The intervening mucosa appeared to be quite normal (*Fig.* 677).

The mesentery in relation to the mucosal tumour masses showed innumerable cysts. The largest were up

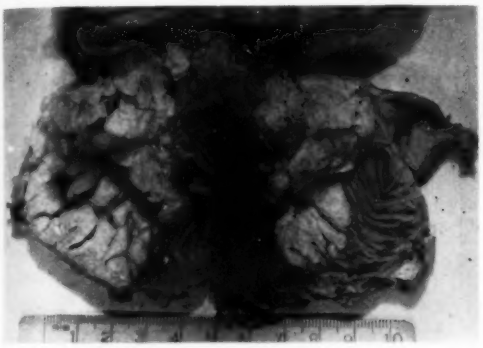

FIG. 677.—*Case* 2. Length of ileum with white, spongy, multicystic tumours projecting into lumen. The intervening mucosa appears quite normal.

to 2 cm. in diameter, while the smaller gave the appearance of honeycombing of the mesentery. In one area in the mesentery of the proximal ileum the cystic area was swollen and showed hæmorrhagic infarction (*Fig.* 678). The cysts appeared to contain clear serous fluid or blood in addition to a small number containing chylous fluid.

Microscopically a distinctive lesion occurred in the submucosal cystic tumours. Here the cystic spaces were often large and irregular and the mucosa was atrophic over much of their surfaces (*Fig.* 679). The muscularis and deeper muscle layers were quite disorganized in the area of the lesion, being represented by irregular muscle strands. The lesions were sharply demarcated from the adjoining gut structures.

The histology of the mesenteric lesion was similar to that in *Case* 1. An additional feature was the hæmorrhagic infarction present in the area near the root of the mesentery. The appearance of honeycombing was particularly striking. The stromal changes of condensed collagenous tissue with very occasional smooth-muscle strands surrounding the cysts, with loose areolar tissue, focal areas of lymphocytic and histiocytic cell infiltration, were similar to the findings in *Case* 1.

DISCUSSION

Aetiology and Pathology.—*Cases* 1 and 2 show extensive lymphangiectasia of the mesenteric tissues. In addition, in *Case* 2 large discrete cystic malformations were present in the ileal mucosal tissues, the intervening gut being quite normal. A congenital origin is supported by the occurrence of isolated mucosal lesions, and by the finding of numerous small mesenteric cysts at laparotomy fifteen years earlier.

FIG. 678.—*Case* 2. Resected area of gut and mesentery showing swellings of mesentery (cut across), with many small cysts and engorged vessels.

FIG. 679.—*Case* 2. Cystic lesion of ileal mucosa. The fibres of the muscularis mucosa are disrupted by the process. There are isolated villi distributed irregularly over the surface of the cysts. Alcian blue and van Gieson. (×51.)

Gross (1953) regards the cysts as being due to growth of congenitally displaced lymphatics which fail to communicate with normal channels. The occurrence of all types of lesion from unilocular single cysts through intermediate forms to a lymphangiectasia is in keeping with this view, as is the predominance of cases in infancy and childhood.

Mesenteric tumours involving elements other than lymphatics may have a similar general origin.

Both the present cases showed an occasional blood-containing cyst, intimately related to the lymphatic cysts, and smooth-muscle strands were observed in the stromal tissues. Conroy and Taniguchi (1951) describe a case of cavernous hæmangioma of the mesentery, while Hartz and van de Stadt's (1949) patient had a large tumour of spongy vascular tissue and irregularly arranged smooth-muscle bundles. Widespread lymphangiectasia affecting other parts of the body is histologically similar to the lesions in the present cases. Lymphangioma of the neck and mediastinum (cystic hygroma) are often multilocular, and form the best recognized group. In the lungs a remarkable diffuse lymphangiectasia may cause death in the neonatal period (Laurence, 1955).

The effect of stasis or lymphatic obstruction in pathogenesis is not easy to evaluate. Mechanical obstruction may produce rapid cystic dilatation of lymphatics as may be seen in the spread of malignant disease, and proliferative changes are seen in the connective tissues about the cysts which may contribute to their increase in size. In *Cases* 1 and 2 inflammatory and proliferative changes could not be related to any particular grouping of the cysts. It seems likely that obstructive factors cause secondary enlargements of cysts and malformations already present and predispose to a vicious circle of vascular thrombosis and regional infarction with further inflammatory obstruction.

The microscopical features of mesenteric cysts and their similarity to lymphangiectasia elsewhere in the body is quite in accord with the view that single unilocular and multilocular lymphatic cysts, multiple simple cysts, and lymphangiectatic tumours represent a common pathological process of maldevelopment, and that angiomatous, muscular, and other elements may rarely be involved in related processes.

Clinical Features.—The present cases were similar in showing a history of several attacks of abdominal colic and vomiting, with complete normality between the attacks. The cysts were palpated in *Case* 1 only, and then were appreciated on only one or two occasions despite repeated examinations. In many of the reported cases no tumour was palpable. This is frequently the case when multiple small cysts are present, and when the cysts are soft and easily compressed. The presence of a gasless shadow on radiological examination of the abdomen may suggest the diagnosis, and there may be demonstrable displacement of the gut near the area.

It seems likely that any extensive lymphangiectasia of the mesentery will result in symptoms during the early decades. Multiple lesions are seldom seen as incidental autopsy findings, and even solitary lymphatic cysts are rarely observed. Rayford (1932) reports only a single lymphangioma of the abdomen in 11,500 autopsies. A review of 15,144 biopsy and 1913 autopsy records at St. George's Hospital, London, showed 2 intraperitoneal lymphangiomata. Both arose from the peritoneum of the lesser sac.

Infrequent presenting symptoms include diarrhœa and the passage of blood in the stools. Miller (1954) reports such a case in a 3-weeks-old infant, and a patient has been recently observed whose chief complaint was diarrhœa with slime in the stools. This 60-year-old woman had had a similar attack of 'colitis' previously. A cystic mass was felt per rectum, and at laparotomy a moderate-sized multilocular lymphoid cyst was present in the mesentery abutting on the sigmoid colon.

Large cysts or solitary cysts are likely to produce acute symptoms due to torsion, rupture into the peritoneum, or hæmorrhage into the cyst. Sudden intestinal obstruction may result from stretching of the gut wall over the cyst.

Acute intestinal obstruction developed in *Case* 1, and several of the reported cases of multiple cystic disease of the mesentery have presented in this way (MacNab and Menzies, 1949, Handelsman and Ravitch, 1954, *Case* 4).

Fever and polymorphonuclear leucocytosis have been additional features of such cases, and mesenteric infarction and surface exudate are frequently present at operation.

The most suggestive clinical features in many of the reported cases are the often repeated attacks of central abdominal colic and vomiting. They may extend over many months with infrequent intervals (as in *Case* 2), or may be short-lived frequent attacks (as in *Case* 1). That the episodes are often accompanied by vomiting suggests that these attacks are due to transient intestinal obstruction. The pain may arise from slight degrees of torsion of the mesentery or to the development of areas of congestive infarction.

SUMMARY

1. Two cases of multiple mesenteric lymphangiomata are reported. They occurred in a boy of 5 years and in a 21-year-old youth. In the latter case the lymphangiomatous state of the mesentery had been observed at laparotomy fifteen years earlier.

2. The aetiology and clinical features of the condition are briefly discussed.

3. Evidence is presented for a congenital origin of mesenteric lymphangiomata. A history of intermittent episodes of transient abdominal colic and vomiting was an important feature of the cases described.

I wish to record my grateful thanks to Mr. T. Rowntree and Mr. S. N. Lytle for their encouragement and permission to publish their cases, and to Mr. J. Wilcox for the photographs.

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RENAL COOLING AND ISCHÆMIA

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THERE has been increasing interest in recent years in such operations as partial nephrectomy, aortic grafting, and renal transplantation. A feature of these operations is the interruption of the blood-flow to the kidney for varying intervals, and it is necessary to consider the effects of temporary ischæmia on subsequent renal function. Cooling the kidney, either locally or by total body hypothermia, could be expected to reduce ischæmic damage as it does in such conditions as incipient gangrene of a limb. It is pertinent, however, to establish the precise value of hypothermia and to determine what functional or anatomical changes may be directly attributable to it. The present study was planned to investigate this problem with a view to establishing a technique of local renal cooling which would allow the blood-supply of a kidney to be arrested for several hours without producing irreparable renal damage.

MATERIALS AND METHODS

Thirty-six healthy, Romney Marsh lambs of both sexes were used in this investigation. After pre-operative urine collections, using a plastic box strapped on the abdomen, the animals were starved for 24 hours. General anæsthesia was induced by intravenous pentobarbital sodium (0·75 gr./10 lb. body-weight) supplemented by open ether on a nasal mask. Under aseptic conditions the left kidney was exposed through a loin incision and completely cleared of perinephric fat. The long left renal pedicle in sheep facilitates delivery of this kidney through the wound. The renal artery and vein were occluded with a rubber-covered Blalock clamp. The ureter was carefully avoided and the clamp tightened rapidly to prevent venous congestion of the kidney. All renal circulation was arrested by this method except possibly a small amount entering along the ureter. A sterile needle thermocouple inserted deeply into the kidney through the hilum was used to measure the intra-renal temperature. After the requisite period of ischæmia the thermocouple was removed and the circulation to the kidney re-established by releasing the clamp. Bleeding from the puncture wound was usually slight. A right nephrectomy was now performed, although in four animals this was delayed for three weeks. Following operation the total urinary output was collected daily for two weeks. Urine was examined for protein and casts and the specific gravity, urea, and electrolyte content were determined. In female animals cystoscopy after intravenous indigo-carmine injection was used to assess renal function. At intervals blood samples were taken for T.N.P.N. and electrolyte determinations. Electrolytes in serum and urine were measured by flame photometry. Animals surviving this procedure were kept under farm conditions for up to twenty weeks, when they were killed and the 'ischæmic' kidneys removed, weighed, and examined histologically. Following fixation in Helly's fluid, hæmatoxylin-eosin, Van Gieson, and Masson's trichrome stains were used.

Local renal cooling was obtained by invaginating the kidney into a sterile plastic bag through which cooled saline was circulated (*Fig.* 680). Oxycyanide of mercury 1–1000 was used to sterilize the bag. By this technique the intrarenal temperature was reduced to levels below 20° C. in approximately ten minutes and held at the required level by varying the temperature of the irrigating fluid. On removing the bag and reopening the renal circulation the kidney temperature returned to normal over a period of approximately five minutes.

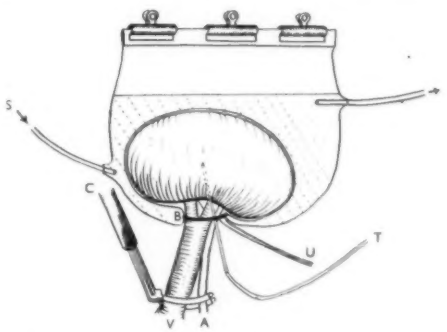

FIG. 680.—Drawing of the cooling technique. The renal vessels (AV) clamped in a Blalock clamp (C). Thermocouple (T) enters the renal hilum and is connected to a galvanometer for recording intrarenal temperature. The plastic bag encloses the kidney and a rubber band (B) holds it in place. Refrigerated saline (S) is run into the bag, which is folded and clipped with paper-clips. The intrarenal temperature can be varied by altering the rate of flow or the temperature of the irrigating saline.

RESULTS

1. Effects of Renal Ischæmia at Body Temperature.—The numbers of animals surviving at four weeks following renal vessel clamping are recorded in *Table I.* Two animals given 1 hour of ischæmia were alive, and had normal T.N.P.N. one month after operation. In 3 animals given 1½ hours of renal ischæmia death occurred within fourteen days. In 4 animals given 2 hours of renal ischæmia death occurred in approximately seven days. The cause of death in all these animals was uræmia. This took the form of increasing lethargy, profuse diarrhœa, and marked loss of weight. Over the terminal period the animals were cast. There was marked hyperventilation with urea frosting of the nose. Blood T.N.P.N. climbed to levels around 300 mg./100 ml. just before death. Serum sodium and potassium estimations remained normal throughout the illness.

Changes in Renal Function.—One hour of renal ischæmia resulted in post-operative oliguria. Urine volume dropped from 300 ml. on the first day to 60 ml. on the seventh day. This was associated with a reduced urea concentration of 1–2 g./100 ml. Some elevation of blood T.N.P.N. occurred to

75 mg./100 ml. in the first week, but at the end of four weeks this had returned to normal. On cystoscopy one week after operation indigo-carmine appeared in good concentration at 7½ minutes.

Urine secretion after 1½ hours of renal ischæmia was reduced to 20–30 ml./24 hours, with anuria ensuing on the third or fourth day after operation.

Table I.—Effect of Varying Periods of Renal Ischæmia at Body Temperature (Opposite Kidney Removed)

Duration of Clamping	*Number of Sheep used*	*Surviving at Four Weeks*	*T.N.P.N. at Four Weeks (mg./100 ml.)*
1 hr.	2	2	44 36
1½ hr.	3	—	—
2 hr.	4	—	—

Two hours of ischæmia resulted in immediate anuria. Any urine secreted was heavily blood-stained and contained large amounts of protein, turning solid on boiling. Indigo-carmine tests in both the 1½-hour and 2-hour 'ischæmic' kidneys failed to reveal any secretion of the dye at cystoscopy one week after operation.

Changes in the Kidney.—Macroscopical and microscopical examination of the kidneys inflicted with 1 hour of ischæmia showed little abnormality when

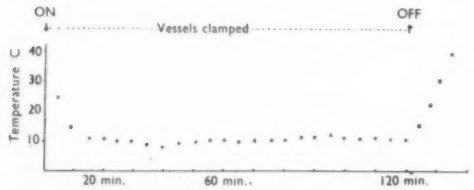

Fig. 681.—Intrarenal temperature record of sheep 141. Average temperature in this case was taken as 11° C. This animal survived and the T.N.P.N. at four weeks was 31 mg./100 ml.

examined four weeks after operation. Some degenerative changes were present in the convoluted tubules, with occasional casts in the collecting tubules.

In the animals dying in uræmia following longer periods of ischæmia, the renal cortex was swollen and pale with congestion of the medulla. The kidneys were soft and friable, with visible infarcts. Microscopically the picture was that of a severe tubular necrosis which was patchy in its distribution. Many tubules were converted into granular hyaline debris (*Fig.* 682). Areas of dilatation of the tubules occurred and many of the collecting tubules contained hyaline and granular casts. The glomeruli appeared surprisingly normal in these kidneys. Little obvious difference was seen between kidneys ischæmic for 1½ hours and those ischæmic for 2 hours except that the changes were more marked in the latter. Capillary engorgement in the boundary zone between cortex and medulla was a feature of the kidneys from uræmic animals.

Recovery from Ischæmic Injury.—In order to assess the degree of recovery from an ischæmic insult of 1½ to 2 hours a series of operations was carried out leaving the opposite kidney intact. Three weeks later the normal kidney was removed, subjecting the damaged kidney to full load. The results of these operations are recorded in *Table II.* One death in uræmia occurred in each group. Renal function in the survivors proved interesting. Following contralateral nephrectomy marked polyuria occurred from the damaged kidney with urine of low specific gravity

Table II.—Effect of Ischæmia at Body Temperature. Contralateral Nephrectomy at Three Weeks

Duration of Clamping	*Number of Animals*	*Number surviving Four Weeks after Contralateral Nephrectomy*	*T.N.P.N. Four Weeks after Contralateral Nephrectomy (mg./100 ml.)*
1½ hr.	2	1	37
2 hr.	2	1	48

(1010) and low urea concentration (1–2 g./100 ml.). Gradual improvement occurred over the next few weeks. Blood T.N.P.N. rose to levels around 100 mg./100 ml. on the fifth day, but in the survivors had returned almost to normal in four weeks. Histological examination of these kidneys revealed patchy tubular necrosis with heavy leucocytic infiltration. In the survivors these areas of necrosis were focal in distribution, with areas of normal tubules in between. No definite histological evidence of regenerating tubules was seen.

2. Effects of Renal Ischæmia with Local Cooling.—In one animal the kidney was cooled for 2 hours using the plastic bag technique to a temperature of 20° C. without interruption of the renal circulation. The opposite kidney was then removed. Recovery following this procedure was uneventful. One month later the blood T.N.P.N. was normal and histological examination of the kidney showed no abnormality. In 18 animals a period of 2 hours of ischæmia was carried out at temperatures ranging from 6° C. to 25° C. In each case the renal pedicle was clamped and the bag was then adjusted. Progressive reduction in renal temperature was produced and after 20 minutes stabilization occurred. The average temperature was taken from considering the temperature curve between 20 minutes and 120 minutes (*Fig.* 681). The number of animals surviving this procedure are recorded in *Table III*, where it can be seen that the temperatures between 10° C. and 20° C. were more effective in preventing ischæmic renal damage. Above 20° C. 2 animals died in uræmia. Between 10° C. and 20° C. only 1 death occurred in 10 animals. Below 10° C. 2 deaths occurred in uræmia, but in 1 of these animals hæmorrhage occurred at the site of the thermocouple needle and this kidney was anuric post-operatively.

Changes in Renal Function.—Animals surviving ischæmia of 2 hours with local hypothermia passed normal daily urine volumes. Over the following two weeks specific gravity and urea content of this urine was normal. No proteinuria occurred.

Excretion of electrolytes by these kidneys was normal. Indigo-carmine excretion by the cooled kidneys occurred in good concentration in 3½–6 minutes when observed at cystoscopy one week after operation. Blood T.N.P.N. showed some slight elevation to 60–90 mg. about the fourth or fifth day after operation, but returned to normal within two to

three weeks. This elevation in T.N.P.N. was comparable with that found after 1 hour of renal ischæmia at body temperature. Sodium and potassium levels in the serum remained unchanged throughout. Blood-pressure recordings in animals allowed to survive 16–20 weeks were normal.

Changes in the Kidney.—Macroscopical examination of the kidneys removed from survivors four

Table III.—Effect of Two Hours' Ischæmia combined with Local Cooling (Opposite Kidney removed)

Average Temperature during Clamping*	*Number of Sheep used*	*Surviving at Four Weeks*	*T.N.P.N. at Four Weeks (mg./100 ml.)*
38° C. (no cooling)	4	—	—
25° C.–20° C.	3	1	26
20° C.–15° C.	6	5	162 50 35 36 29
15° C.–10° C.	4	4	31 36 30 39
10° C.– 5° C.	5	3	42 33 43

* The average temperature was determined from 5-min. records of intrarenal temperature. Since approximately 20 min. elapsed before renal temperatures stabilized, this period was not included in calculating the average temperature.

weeks after operation showed no abnormality apart from the site of the thermocouple needle puncture. In animals allowed to survive up to twenty weeks post-operatively the kidneys had undergone compensatory hypertrophy. Histological examination of these

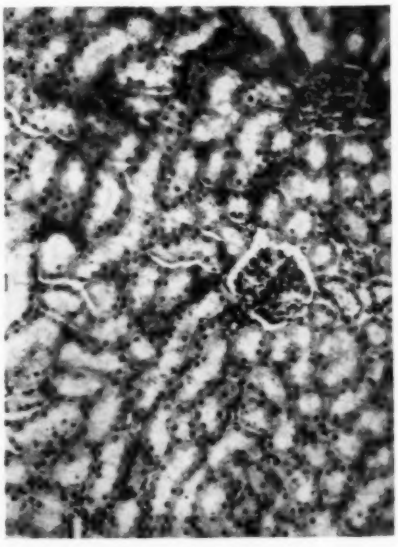

Fig. 683.—Photomicrograph of kidney of sheep 137. Two hours of ischæmia at 20° C. Animal killed at four weeks. T.N.P.N. 38 mg. 100 ml. Normal histological appearance. H. and E. (× 97.)

kidneys showed a completely normal pattern at four weeks after operation, with no evidence of fibrosis or tubular damage (*Fig.* 683). Examination of kidneys from animals dying in uræmia following ischæmia at temperatures above 20° C. showed typical ischæmic injury, with necrosis of the convoluted tubules. In 1 animal dying in uræmia twenty-five days after

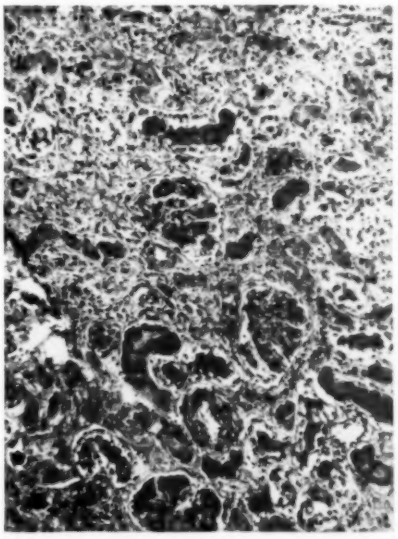

Fig. 682.—Photomicrograph of kidney of sheep 113. Two hours of ischæmia at body temperature. Animal died eleven days later in uræmia. Note extensive tubular necrosis. Glomeruli relatively normal. H. and E. (× 97.)

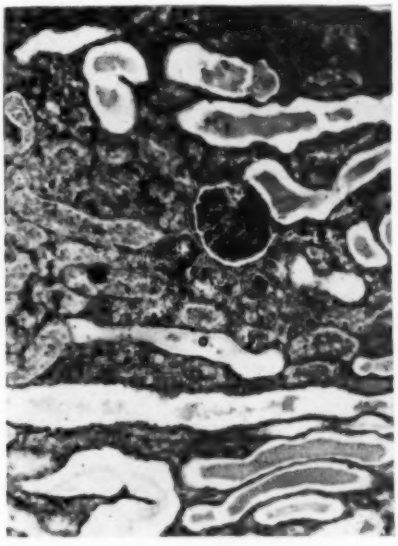

Fig. 684.—Photomicrograph of kidney of sheep 134. Two hours of ischæmia at 19° C. Animal died twenty-five days later in uræmia. Note the cystic dilatation of tubules. H. and E. (× 97.)

operation at 19° C. markedly dilated tubules were seen lined by flattened epithelium (*Fig.* 684). This appearance was seen also in 1 animal dying thirteen days after operation at 6° C.

DISCUSSION

In the light of previous work it appears that nephrectomy on one side accompanied by occlusion of the renal vessels on the other is accompanied by impaired renal function due to tubular injury. This is reflected by fall in urea clearance and progressive rise in T.N.P.N. The tolerance to ischæmia appears to vary with species of animal. In rabbits, under these conditions, 1 hour of ischæmia has a mortality of 20 per cent whereas 2 hours is uniformly fatal (Badenoch and Darmady, 1947). In the dog, however, 2 hours is tolerated without mortality (Hamilton, Phillips, and Hiller, 1948); 3 hours results in survival in the majority, 4 hours gives a mortality of 50 per cent, and 6 hours is uniformly fatal (Van Slyke, Phillips, Hamilton, Archibald, Dole, and Emerson, 1944). In the rat Koletsky (1954) has studied functional and histological changes following ischæmia. No previous results are available in the sheep. The results recorded here in sheep subjected to renal ischæmia under normothermic conditions show that beyond one hour the resultant renal damage rendered the kidney unable to support the animal when the opposite kidney had been removed. This damage was mainly in the convoluted tubules.

The literature on hypothermia and renal function is meagre. Bickford and Winton (1937), working with isolated perfused kidneys, described alteration of urinary secretion with falling temperature. The 'cold' kidney passed urine approximating to glomerular filtrate, indicating arrest of tubular function. This effect was maximal at 10° C. and they claimed that recovery occurred on warming. Using total body hypothermia Ross (1954) found that urine production was diminished with cooling to 25° C. Little or no urine was produced. Nedzel (1952) claims to have demonstrated the operation of the Trueta renal shunt in dogs subjected to body chilling. This gave rise to vasoconstriction in the renal cortex, with cessation of the flow of urine. One observation in these experiments may support this contention. Normally on releasing the clamp on the renal pedicle, after ischæmia at body temperatures, the kidney swelled rapidly and became extremely tense. The surface changed from a dusky purple to a bright pink as the circulation returned. In chilled kidneys such rapid return of circulation did not occur. The surface remained blue, with punctate areas of erythema developing relatively slowly. Several explanations of this phenomenon are possible. Either there was persistent cortical ischæmia produced by vasoconstriction in the cortical vessels of the chilled kidney or alternatively under hypothermic conditions the concentration of vasodilating metabolites in the ischæmic kidney was markedly reduced. Further, under hypothermic conditions, spasm throughout the whole renal arterial tree may be more marked.

Angiographic studies on hypothermic kidneys would certainly be of interest in this connexion. Miles and Churchill-Davidson (1955) have measured P.A.H. and inulin clearances in dogs cooled to 22° C. by surface cooling. They claim that the mean effective renal flow dropped to half normal at 29° C. and one-quarter normal at 24° C. This paralleled changes in cardiac output and they draw no conclusions as to the state of the renal vessels at these temperatures. The effects of total body hypothermia to 26·7 C. on ischæmic renal damage is reported by Moyer, Heider, Morris, and Handley (1957). They remark that this temperature will at least double the period of ischæmic tolerance, but in their operations the exact intrarenal temperature was not recorded.

The results summarized in *Table III* show that there was a definite increase in tolerance to ischæmia under local hypothermia and that this effect became more important at temperatures below 20° C. In order to achieve this temperature some technique of local renal cooling is necessary, since total body cooling to these temperatures is unsafe. Semb (1956) reports experiments on dogs using a similar method of local renal cooling, and found that under such conditions dogs would survive periods of ischæmia of up to 6 hours. There appears to be a greater period of ischæmia tolerance in the dog kidney under hypothermic conditions than the results here would suggest in the sheep.

In attempting to prolong tolerance to ischæmia by hypothermia, deleterious effects as a result of low temperatures must be considered. Knocker (1955) described lipoid accumulations in the kidney after body cooling. This worker questions "whether the insult imposed upon the tissues by cold is a sound preparation for ensuing anoxia". In the present study no early biopsies were carried out after ischæmia and hypothermia, but no adverse effects attributable to hypothermia have been detected in the kidneys examined four weeks after operation. The excellent post-operative function of the hypothermic kidneys suggests that any changes are rapidly reversible. The effect of hypothermia on the perinephric tissues was noteworthy. At the conclusion of the operation the perinephric fat which lay in contact with the bag was hard and lardaceous in appearance. Examination of this area at subsequent autopsy invariably revealed areas of fat necrosis and fibrosis, an effect due presumably to the reduction of temperature, since such changes were never seen after operations at body temperature. Wound healing appeared to be retarded in the animals subjected to local renal hypothermia and in 1 case complete wound breakdown occurred on the thirteenth day after operation. Retardation of wound healing and increased risk of wound sepsis appeared to be the only detrimental results attributable to the hypothermic technique.

The results with temperatures below 10° C. indicate that functional efficiency in the kidney was impaired. In order to reduce intrarenal temperatures below 10° C. by surface cooling a much lower temperature must obtain in the renal cortex. Such low temperatures caused hardening of the kidney surface, apparently due to solidification of intracellular fat, and cortical necrosis ensued. In a series of renal transplants cooled by a perfusion technique to temperatures approaching 0° C. cortical necrosis was found, and at the present moment it would appear unwise to reduce renal temperature below 10° C. in human operations. At temperatures between 10° C. and 20° C. the technique reported here would have definite advantages in certain renal procedures such as partial nephrectomy, renal artery replacement, and certain cases of nephrolithotomy.

SUMMARY

A technique of local renal cooling is described. By this means renal damage from ischæmia can be reduced. The most effective temperature lies between 10° and 20° C. No permanent harmful effects on renal function follow cooling to these temperatures and the method is suggested as of possible value in the conservative surgery of the kidney.

Acknowledgements.—The work reported in this paper was assisted by a grant from the Medical Research Council of New Zealand. Part of it is incorporated in a thesis for the degree of Ch.M. of the University of New Zealand. I wish to acknowledge the helpful advice and criticism of Professor M. F. A. Woodruff, in whose Department this work was carried out. My thanks are also recorded to Dr. J. G. Llaurado for the electrolyte estimations; to the Department of Photography, Otago Medical School, for photographs; and to Mr. John Flawn for his technical assistance.

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CHRONIC THYROIDITIS FOLLOWING THYROTOXICOSIS

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THE various forms of thyroiditis—De Quervain's, Hashimoto's, and Riedel's types—are generally considered to be clinically and pathologically distinct from each other and from primary hyperplasia of the thyroid. The majority of observers are in agreement with Joll (1939), who consistently failed to find evidence of preceding hyperthyroidism in his detailed study of 81 cases of struma lymphomatosa. There is, however, a considerable minority in favour of the opposing view—namely, that thyroid hyperplasia and thyroiditis are aetiologically related. The evidence has been largely indirect, and has often depended on clinical impressions and histories unsupported by pathological findings. Those instances in which the same patient is subjected to thyroidectomy on more than one occasion provide an opportunity of testing the possibility of progression from thyroid hyperplasia to thyroiditis.

Two cases have recently been seen in which thyroiditis occurred many years after partial thyroidectomy for toxic diffuse goitre.

CASE REPORTS

Case 1.—A married woman, born in 1907, underwent a partial thyroidectomy in 1941 for toxic goitre. Symptoms of thyrotoxicosis had been present for about a year; there was slight exophthalmos, and the basal metabolic rate was plus 65 per cent. Histological examination of the excised tissue showed diffuse epithelial hyperplasia and colloid depletion, accompanied by a slight degree of focal round-cell infiltration.

She remained well for about nine years, but thereafter symptoms of thyrotoxicosis steadily returned, and thiouracil treatment was begun in 1952 and continued for eighteen months. The goitre increased markedly during the summer of 1956. On examination in September, 1956, the thyroid enlargement was much more pronounced in the right lobe. Tremor and tachycardia were of slight degree, and there was no exophthalmos. There had been slight loss of weight. Radiography showed some compression and deviation of the trachea to the left. Hoarseness and irritating cough were the most prominent symptoms.

At operation in November, 1956, there was gross fibrosis of the infrahyoid musculature, and the thyroid was very adherent to the trachea. The right lobe of the thyroid was excised with considerable difficulty, leaving behind a thick posterior layer which did not appear to contain thyroid tissue.

Post-operatively the patient was well and she gained weight. One year later she developed signs of mild hypothyroidism, which responded to 1 gr. of thyroid extract daily.

PATHOLOGY.—The excised lobe measured 7·5 × 5 × 4 cm., and weighed 98 g. It was smooth, firm, greyish-white, and like a lymph-node in consistency. The cut surface was uniformly greyish-white, with a few small cysts but no obvious nodular structure. Muscle-fibres were attached along one margin.

Microscopically, the outstanding features were degenerative changes in the thyroid follicles and an intense diffuse plasma-cell infiltration (*Fig.* 687). The follicles were small or medium-sized; many were totally collapsed, and in some areas groups of epithelial cells were so intermingled with plasma cells that no follicular structure was recognizable. In many areas the epithelial cells, whether forming the lining of follicles or arranged in solid clumps, were of large oxyphilic type. Elsewhere the follicles were lined by neutrophilic cuboidal or low columnar epithelium, with a few ill-defined nodules showing hyperplasia of the degree associated with exophthalmic goitre. Only a minority of the follicles contained colloid; the remainder were empty or were occupied by multinucleate giant cells (*Fig.* 688).

Diffuse plasma-cell infiltration involved all parts of the lobe, though varying in intensity from one area to another. The comparatively few lymphocytes showed a follicular arrangement, often with germinal centres. Polymorphs

were not present, nor were there any extrafollicular giant cells. Examination by polarized light did not reveal any anisotropic material.

The small cysts were empty; they were lined by a single layer of flattened epithelium, and old and recent hæmorrhage was seen in their walls.

giant cells. Fibrosis was of about the same degree as in the later specimen.

Thus the same elements made up the histological picture in both specimens, though the first was typical of exophthalmic goitre, while the second showed an undoubted chronic thyroiditis.

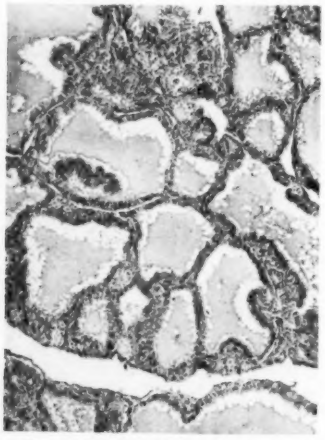

FIG. 685.—*Case* 1. Diffuse toxic goitre in 1941. (All hæmatoxylin and eosin.) (× 80.)

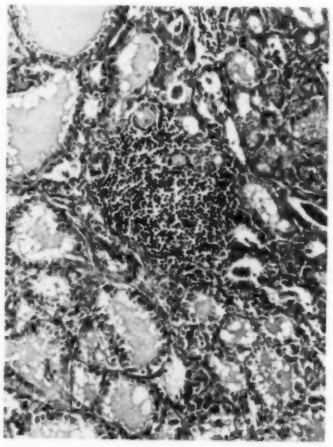

FIG. 686.—*Case* 1 (1941). A focus of lymphocytic and plasma-cell infiltration in toxic goitre. Giant cells are seen in some of the thyroid follicles.

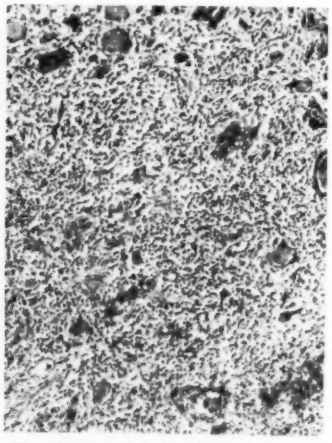

FIG. 687.—*Case* 1. Chronic thyroiditis in 1956. Intense diffuse plasma-cell infiltration, and disintegration of the thyroid follicles. Some of the epithelial cells are of large oxyphilic type.

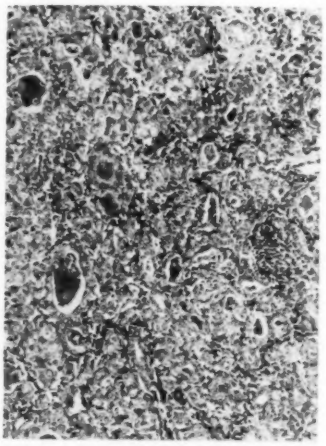

FIG. 688.—*Case* 1 (1956). An area in which the follicles are well preserved. Some contain colloid, others syncytial giant cells.

Fibrosis of the gland was of slight degree, insufficient to produce a definite nodularity.

The muscle-fibres attached to the thyroid showed a sparse lymphocytic and plasma-cell infiltrate.

Re-examination of the earlier (1941) specimen showed the picture of diffuse epithelial hyperplasia with focal 'thyroiditis' (*Figs.* 685, 686). The sparse inflammatory foci were composed of lymphocytes, occasionally in the form of lymphoid follicles, and a few plasma cells. These foci were associated with collapse of the thyroid follicles, oxyphilic change in the epithelium, and intrafollicular

Case 2.—A married woman, born in 1917, had been aware of a swelling in the neck since the age of 10. At the age of 19 she had developed symptoms of hyperthyroidism, with a large symmetrical goitre, exophthalmos, and a basal metabolic rate of plus 66 per cent. Subtotal thyroidectomy in 1937 had been followed by a period of good health, apart from transient loss of voice. The excised gland was not examined histologically. On re-examination in 1947 she appeared myxœdematous; the basal metabolic rate was minus 25 per cent, and the serum-cholesterol 222 mg. per cent; she was treated with thyroid

extract. One year later, a diagnosis of 'recurrent thyrotoxicosis' was made, and she was treated with thiouracil for one month, but subsequently thyroid medication was resumed. Enlargement of the thyroid re-appeared in 1950, and pressure symptoms appeared in 1956. On examination in February, 1957, she was pale and overweight, but not definitely myxœdematous. There appeared to be a cyst in the thyroid isthmus. The B.M.R. was minus 33 per cent, and the serum-cholesterol 205 mg. per cent. X-ray examination showed compression and some deviation of the trachea to the left. At operation in July, 1957, there was considerable fibrosis around the thyroid; the enlargement was principally in the right lobe and isthmus, most of which were resected.

PATHOLOGY.—The specimen comprised two irregular lobulated pieces of tissue, measuring 5 and 3·5 cm. in their greatest diameters, as well as four smaller fragments, weighing in all 22 g. The cut surface was pale grey throughout, with no obvious cysts or nodules, but showing a rather ill-defined lobulation.

The microscopical picture was a combination of epithelial hyperplasia, inflammatory cellular infiltration, and fibrosis.

The thyroid follicles were small, lined by cuboidal or low columnar epithelium, and their colloid content was poor. In some areas the tall columnar epithelium, irregular outline of the follicles, and scalloping of the colloid provided the appearances of primary thyroid hyperplasia. In other areas the follicular structure was disorganized, and the lumina scarcely recognizable. Some acini were lined by large oxyphilic cells, and there was one focus of squamous metaplasia.

The inflammatory infiltrate was composed mostly of plasma cells, though lymphocytes were also present, sometimes in the form of lymphoid follicles with germinal centres. Cellular infiltration was very dense at the periphery of the gland and at the periphery of individual lobules; elsewhere it varied greatly in density, and was nowhere entirely absent.

The gland was divided into irregular nodules of varying size by bands of dense fibrous tissue. One of the nodules showed the trabecular arrangement suggestive of true adenoma.

DISCUSSION

It is not proposed to discuss in detail the possible diagnostic labels which might be applied to the second operation specimen in these 2 cases. While some would readily diagnose them as struma lymphomatosa, or as a variant of this condition, others employing stricter criteria would point to the paucity of the lymphoid follicles and the limited extent of the oxyphilic change, and assign them to the category of chronic non-specific thyroiditis. For the present purpose it seems sufficient to use the descriptive and non-committal term 'plasma-cell thyroiditis', the more so as there is good reason to believe that the finer histological distinctions between the various forms of chronic thyroiditis are without clinical significance (Peterson and Shidler, 1957).

The chief interest of these 2 cases lies in the apparent progression, over a period of many years, from primary diffuse hyperplasia to chronic thyroiditis. The possibility of two unrelated, and not uncommon, thyroid disorders affecting the same patient at different times is unlikely, both in view of the clinical course and on morphological grounds.

The idea that epithelial hyperplasia might act as the initiating factor in Hashimoto's disease was suggested as early as 1931, when Graham and McCullagh, commenting on the lack of a history of hyperthyroidism in their own and in Hashimoto's original cases, remarked that were it permissible to assume an antecedent hyperthyroidism, an explanation of pathogenesis would not be difficult. One can readily picture the focal thyroiditis which so commonly accompanies primary thyroid hyperplasia becoming transformed into the generalized inflammatory reaction characteristic of chronic thyroiditis. It was suggested by Vaux (1938) that lymphadenoid goitre should be regarded as excessive involution of the thyroid following mild thyrotoxicosis, and Goldberg and Davson (1948) considered that "a toxic phase is sufficiently frequent to count as an important factor in pathogenesis". Goetsch and Kamner (1955) traced a continuous pathological process from toxic diffuse goitre through chronic thyroiditis to Riedel's struma, and Chesky, Dreese, and Hellwig (1951) went further in including subacute thyroiditis (usually regarded as an infective lesion) as a stage in the sequence. They maintain that "there are only quantitative differences between the severe grades of chronic thyroiditis (Riedel's, Hashimoto's and De Quervain's type) and localized foci of chronic thyroiditis in exophthalmic goiter and nodular goiter. Both show the same morphologic characteristics, the only difference being in intensity." In the same vein, Levitt (1954, 1957) described a series of six phases from epithelial hyperplasia through Hashimoto's disease to Riedel's fibrosis, but he was careful to distinguish chronic thyroiditis as an inflammatory condition unrelated to the "life history of the toxic thyroid gland".

None of these authors described actual progression in the same patient from hyperplasia to chronic thyroiditis. Of the 22 patients in Levitt's series who underwent thyroidectomy twice, 16 showed progression and 4 retrogression; of those who progressed, only one advanced by as much as two of his six arbitrary phases. Goetsch and Kamner's cases each had only one thyroidectomy; their support for the progression theory was based on a grouping of their cases in such a way as to show an orderly sequence from hyperplasia to thyroiditis, and on eliciting a previous history suggestive of hyperthyroidism. Chesky and others (1951), from a study of supravital and paraffin preparations in 169 cases of chronic thyroiditis, considered that the sequence of events was as follows: Macrophages enter the follicular lumen and ingest the colloid; the follicle collapses, and the macrophages fuse to form a syncytium; the macrophages leave the lumen, become stranded in the interfollicular stroma, and degenerate; the liberated colloid then attracts lymphocytes by chemotaxis. They concluded that degeneration of the follicular epithelium is the final stage in the process, due to accumulation of lymphocytes around and between the follicles, and not the initial stage, as is more generally supposed. They believed that excess of thyrotropic hormone is the most important factor in causing colloid changes responsible for the attraction of macrophages.

It is therefore rather surprising that most histological studies on patients who have had more than one thyroidectomy (summarized by Hazard, 1955) have failed to show progression of the inflammatory process, or progression from focal to diffuse thyroiditis (Harland and Frantz, 1956). The case reported by Scarcello and Goodale (1941), with a 13-year

interval between thyroidectomy and autopsy, was thought to represent progression from primary hyperplasia to Hashimoto's disease, but the latter diagnosis was by no means established histologically, and seems unlikely from the clinical description. Friedman (1949) mentioned having seen a number of cases in which the residual tissue after subtotal resection for hyperthyroidism showed a pattern indistinguishable from that of Hashimoto's disease, and illustrated one case in which the third operation specimen showed such a picture. Single cases have been reported of progression from Graves' disease to a picture resembling Riedel's struma (Boyden, Coller, and Bugher, 1935), and from an early to a later stage of lymphadenoid goitre (Rabson and Arata, 1949).

The most extensive study is that of Spjut, Warren, and Ackerman (1957), who collected 71 cases of recurrent goitre in which two or more specimens of thyroid were available, as well as 5 cases in which the residual thyroid tissue was examined at autopsy. The initial diagnoses were Graves' disease, toxic (non-exophthalmic) goitre, and non-toxic nodular goitre. Their findings make it clear that in the great majority of cases there is no progression of the lesion as judged from a comparison of the first with subsequent thyroidectomy or autopsy specimens. Only one of their cases (in the non-toxic group) progressed to a condition which conformed to their criteria for struma lymphomatosa. Seven other cases, of which four were in the toxic groups, progressed to a condition resembling struma lymphomatosa, and were regarded as "near misses". These they interpreted as exhaustion atrophy rather than chronic thyroiditis.

The two cases reported here appear to represent true examples of progression from hyperplasia to thyroiditis. A comparison of the first and second specimens from *Case* 1 bears out Goetsch and Kamner's view that the difference between the two conditions is only a quantitative one. The hyperplasia which is the outstanding feature of the first specimen is readily recognizable in the second. The plasma-cell infiltration which is so intense in the second specimen is present, though focal and of mild degree, in the first. Oxyphilic cells, being associated with inflammatory changes, are sparse in the first specimen and numerous in the second. Intrafollicular giant cells are common to both specimens. Fibrosis is also present in both. Indeed, perithyroiditis is the only histological feature of the second specimen whose origins cannot be traced in the first.

It is unfortunate that in *Case* 2 the earlier specimen is not available for study. The typical clinical picture of exophthalmic goitre, however, leaves little doubt as to the histological structure of the thyroid on the earlier occasion. Progression in this case is less advanced than in *Case* 1, as in the second specimen epithelial hyperplasia is still prominent, and plasma-cell infiltration, though intense in some areas, is comparatively mild in others.

It is evident from the rarity of such reports that these cases are exceptions to the general rule. If chronic thyroiditis is indeed preceded by epithelial hyperplasia, it may well be that the hyperplasia occurs in a form unassociated with excessive production of thyroid hormone (Lindsay and others, 1952), perhaps occurring as a consequence of decline in ovarian function (Parmley and Hellwig, 1946), or as a compensatory phenomenon in 'primary thyroid failure' (Skillern, Crile, McCullagh, Hazard, Lewis, and Brown, 1956).

SUMMARY

Histological evidence suggests that chronic thyroiditis is preceded by epithelial hyperplasia, but only rarely is progression from thyrotoxicosis to chronic thyroiditis demonstrated in the same patient.

Two cases are reported in which toxic diffuse goitre was followed, at intervals of 15 and 20 years, by plasma-cell thyroiditis. In one case, both thyroidectomy specimens were available for study, and the origins of the thyroiditis could be traced in the histological picture of the earlier specimen.

I am indebted to Mr. A. Nicholson and Mr. S. Rose for permission to publish their cases; to Dr. L. Stent and Professor A. C. P. Campbell for criticism; and to N. Mowat, F.I.M.L.T., for the photomicrographs.

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PNEUMATOSIS CYSTOIDES INTESTINALIS

By J. H. SHOESMITH and W. P. CRONE, Leeds

Intestinal pneumatosis is an uncommon disease in man, but has been identified more frequently in animals, particularly swine. It is characterized by the presence of single or multiple gas-filled cysts of various sizes up to several centimetres in diameter. These cysts are most commonly found in the small intestine, are rare in the stomach, omentum, and large intestine, and are particularly uncommon in the descending and sigmoid colon. They may be situated in the submucosal or the subserosal layers of the intestine and usually contain an odourless, non-inflammable gas, the predominant constituents being nitrogen 70–90 per cent, carbon dioxide, and oxygen; on occasions hydrogen and methane have been found in small amounts.

Most of the reported cases have been in adults, particularly men between the ages of 30 and 50 years, but MacKenzie (1951) reported 12 cases in infants, as well as finding over 30 cases in the literature occurring in childhood.

Microscopically the intestinal mucosa appears normal and unbroken, with gas-containing submucosal or subserous sacs lined by flattened or cuboidal cells and often some giant cells.

The theories of causation have been well reviewed by Matthews (1954), and the commonly accepted theory is that of Masson (1925), who studied the condition in swine. He believed that the cysts were due to dilatation of lymphatic channels following lymph stagnation in tortuous and obstructed channels. He also thought that carbon dioxide and lactic acid were absorbed from the bowel into the blocked lymphatics and that this was followed by liberation of carbon dioxide and by diffusion of nitrogen, oxygen, and occasionally methane and hydrogen from the capillaries.

It is difficult to attribute a definite clinical picture to the presence of multiple gas cysts in the walls of the intestines. Usually symptoms occur because of some associated or concurrent gastro-intestinal disease, the most common reported one being a stenosing ulcer of the pylorus and also intestinal tuberculous ulceration and gastric carcinoma. Symptoms may also occur from complications of the disease. Intestinal obstruction following blockage of the lumen by a mass of cysts, volvulus, and intussusception has been recorded. Radiologically, gas may be seen under the diaphragm owing to spontaneous rupture of a cyst.

The presence of free air in the peritoneal cavity may occur from a number of causes, but if gas under the diaphragm is observed over a prolonged period in the absence of a perforated viscus, pneumatosis cystoides intestinalis should be considered because it is the only known condition which can explain a prolonged or recurrent pneumoperitoneum.

The first person to report this disease was probably Duvernoy (1738), but the first full account is attributed to Bang (1876), who found this condition at necropsy. Hahn (1899) first reported the condition in a living subject, and since then, despite its early recognition, Schorr, Ullmann, and Laufer (1951) and Koss (1952), who have extensively reviewed the literature, have only found about 250 cases.

In 1949 Gazin, Brooke, Lerner, and Price reported the first pre-operative radiological diagnosis, and more recently cases involving the sigmoid colon have been reported by Matthews (1954), McGee, Penny, and Williamson (1956), and Griffiths (1955).

Two further cases of intestinal pneumatosis, both involving the sigmoid colon, are now described.

CASE REPORTS

Case 1.—A man aged 50 years was referred to out-patients with a diagnosis of carcinoma of the rectum. He gave a six-months' history of intermittent diarrhœa, each attack of diarrhœa lasting from two to three weeks, followed by normal stools for a similar period. During the attacks he would pass up to six watery stools a day mixed with slime, and on two occasions he had noticed blood in small quantities. The attacks were associated with colicky lower abdominal pain which was not severe.

Fig. 689.—Large intestine, including anus and rectum, showing numerous sessile hemispherical tumours.

He had lost 1½ st. in weight, had a poor appetite, and felt lethargic. For ten years he had suffered from winter bronchitis.

Rectal examination revealed a large polypoid mass situated on the posterior wall of the rectum. He was admitted to hospital with a diagnosis of carcinoma of the rectum.

A radiograph of the chest was normal and the hæmoglobin 100 per cent. A biopsy of the rectal tumour was performed through a proctoscope and the histology reported showed chronic inflammatory changes.

However, in view of the history and despite a negative biopsy, an abdominoperineal resection was carried out.

His convalescence was satisfactory and he was discharged from hospital free from symptoms. When seen one year later he had had no bowel symptoms and was gaining weight.

Pathological Report (W. G.).—Forty cm. of large intestine, including anus and rectum (*Fig.* 689). The whole of the intestine up to and including the rectum shows numerous sessile hemispherical tumours measuring up to 4 cm. in diameter. Some of these are coalescing to form larger masses and are yellowish in colour. On section they consist of empty cystic spaces with thin walls.

Histology: They are empty cysts covered by normal mucosa and submucosa. They are lined mainly by flattened cells, but in some areas by cuboidal cells. This is a beautiful example of intestinal pneumatosis.

Case 2.—A man aged 60 years was first seen complaining of diarrhœa three to four times daily and lower abdominal distension for six months. The stools

were very liquid and light coloured and initially blood-stained. Flatulence was a marked feature and he also complained of swelling of the left ankle in the evening which disappeared by morning.

On examination there was marked distension of the flanks and mid-abdomen. Digital examination revealed a slightly enlarged prostate and 'ballooning' of the rectum.

FIG. 690.—Barium enema showing multiple small filling defects in the descending and proximal portion of the sigmoid colon.

Considerable ankle œdema was present with no joint abnormality.

A barium enema showed multiple small filling defects present in the descending and proximal portion of the sigmoid colon (*Fig.* 690). The length of the colon involved was estimated at about 2 ft. and a tentative diagnosis of multiple polyposis, probably with malignant change, was made. The blood-picture was normal.

To elicit a diagnosis, a laparotomy was performed through a left lower paramedian incision. The descending and pelvic portions of the colon were greatly thickened by the presence of innumerable small swellings within the bowel wall. Many of these were clearly visible. The swellings extended from the last 2 in. of the transverse colon to the lower sigmoid colon, and were absent throughout the rest of the intestines.

The diagnosis of pneumatosis cystoides intestinalis was made at this stage and excision of the involved segment of the colon carried out with an end-to-end anastomosis.

Convalescence was uneventful and the patient was discharged seventeen days later, free from symptoms and with normal bowel action.

Pathological Report (I. D.).—Forty-five cm. of large intestine showing broad and exaggerated mucosal folds, especially in the distal 15 cm. On the cut surface throughout the specimen can be seen small submucosal cysts which vary in size from a pinhead to 1 cm. in diameter (*Fig.* 691). No subserosal cysts are present.

Histology: The mucosa is healthy. In the submucosa between the muscularis mucosa and the muscle coats are a number of cystic spaces which are lined by an almost

FIG. 691.—Cut surface of the specimen showing small submucosal cysts.

continuous membrane of giant cells which lie on an outer thin coat of granulation and fibrous tissue. Patchy eosinophilic reaction is present in places.

CONCLUSIONS

These two cases of pneumatosis cystoides intestinalis are of interest and importance because the main presenting symptom in each case was attacks of blood-stained diarrhœa associated with lower abdominal pain. Also, in *Case* 1, the gas cysts involved the rectum as far as the anal margin and involvement of the rectum has only been reported to our knowledge once before, by Griffiths (1955).

We wish to thank Mr. H. S. Shucksmith and Mr. A. J. C. Latchmore for permission to publish these cases, and Dr. W. Goldie and Dr. I. Dawson for the pathological reports.

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AN UNUSUAL FOREGUT ANOMALY

AN EXTRAGASTRIC POUCH COMMUNICATING WITH THE PANCREATIC DUCT

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THE anomaly of the foregut in this patient is of interest because of the mode of presentation and the transient nature of the physical signs, together with the operative, radiological, and pathological findings.

CASE REPORT

In May, 1957, a thin underdeveloped youth, aged 17 years, was admitted to the Medical Unit of the Cardiff Royal Infirmary. His first symptoms began at the age of 9 years. Whilst sitting in school after lunch, he experienced severe upper abdominal pain which lasted for several hours. In the ensuing three months similar attacks occurred. For two years, until he was 11 years old, there was freedom from pain. Since the age of 11 years, for the last six years, it had increased in frequency and severity. From December, 1956, the pain had occurred almost daily, coming on at any time up to 30 min. after a meal, particularly after breakfast or lunch. The composition of the meal did not affect the pain, which lasted from ten minutes to several hours, was localized to the epigastrium, and was not relieved by alkalis. It varied in intensity from a mild discomfort to a severe colic, causing him to roll on his bed. It was not now associated with nausea or vomiting, as was formerly the case. Rarely he was awakened at night with pain, but on these occasions it was never severe. There had been no hæmatemesis or melæna. His appetite was good, but as he knew that food brought on the pain he was afraid to eat. Bowels and micturition were normal.

A diagnosis of phæochromocytoma had been put forward to account for the abdominal pain and a chance finding of a systolic blood-pressure of 150 mm. during an attack of pain. Extensive investigations at two hospitals elsewhere from 1953 to 1956 failed to confirm the diagnosis.

ON EXAMINATION.—He was 4 ft. 11 in. in height and weighed 5 st. 6 lb. There was evidence of delayed puberty. The only abnormal physical sign was tenderness on deep palpation in the epigastrium. Blood-pressure 110/85.

RELEVANT INVESTIGATIONS.—Barium meal and follow-through revealed no abnormality. There was no fæcal occult blood.

PROGRESS.—On May 14, 1957, a smooth, rounded, firm tumour, about 2 in. in diameter, was felt in the epigastrium just to the left of the midline. The position of the lump corresponded exactly with the site of the pain. The tumour was felt only when the pain was present, but could not be felt during every attack.

Antispasmodics had a variable effect on the pain. Amyl nitrite produced immediate relief lasting for 5 min. Ten minims of belladonna relieved the pain in 15 min., its effect lasting all day. Atropine gr. 1/200 intravenously relieved the pain in 2 min. but it recurred 6 min. later.

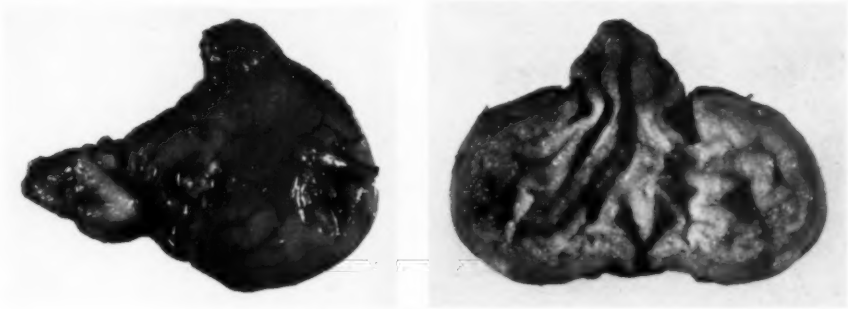

FIG. 692.—Specimen removed at operation (natural size).

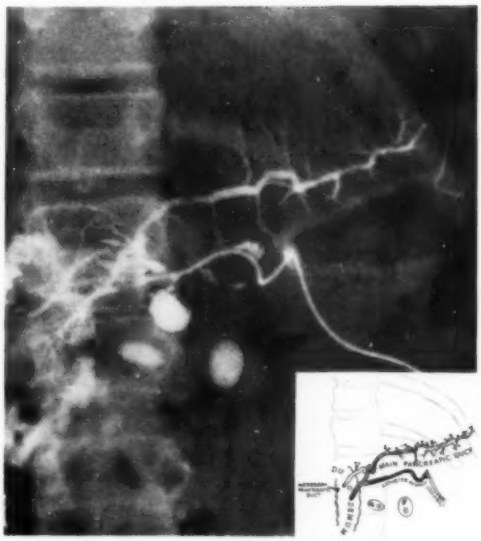

FIG. 693.—Post-operative pancreaticogram and explanatory diagram.

In the light of these findings the diagnosis of an intra-abdominal cyst with a smooth-muscle wall was made, and the patient was referred for surgical treatment.

AT OPERATION (May 29, by Mr. D. B. E. Foster).—An upper midline incision revealed a tumour 3 in. in diameter, thinly veiled by the lesser omentum, which presented above the lesser curve of the stomach. The lesser omentum was divided. The tumour had the appearance of, and felt like, a thick-walled muscular pouch, which contracted spasmodically when it was first

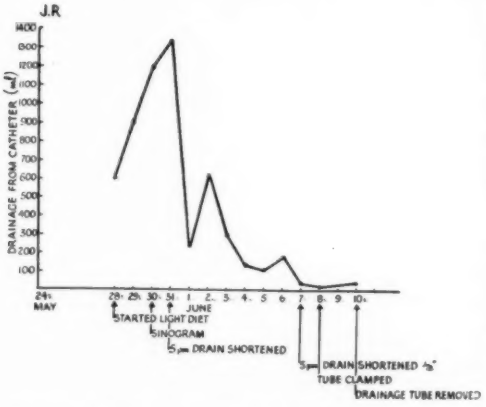

FIG. 694.—Graph to show amount of drainage from the catheter.

touched. Dissection revealed it to be lying on the head of the pancreas, from which many small vessels were found to be passing into it. After the tumour had been mobilized by dividing these vessels, a duct could be seen passing from its deep surface into the head of the pancreas. The tumour was removed by dividing the duct flush with the surface of the pancreas.

A fine rubber catheter was passed into the distal cut end of the duct and sutured in position. The catheter was brought out through a separate incision in the abdominal wall. The main incision was closed in layers.

PATHOLOGY.—On cut section the specimen was a thick-walled muscular sac lined with gastric mucosa, with characteristic rugæ (*Fig.* 692). There was no ulceration of the mucosa. Histological examination confirmed the presence of normal gastric mucosa, but with well-marked hypertrophy of the muscle coats.

POST-OPERATIVE COURSE.—After operation a copious alkaline secretion (*p*H 8·4) poured from the catheter, reaching a maximum of 1340 c.c. on the seventh post-operative day. A sinogram on the sixth post-operative day revealed that the track opened into the main pancreatic duct, before the latter opened into the duodenum (*Fig.* 693). The catheter was withdrawn half an inch from the main pancreatic duct, and the secretion fell by 240 c.c. in the following 24 hr. (*Fig.* 694). The drain was shortened further on the fourteenth post-operative day, clamped the next day with no ill effect, and removed completely seventeen days after operation. The wound healed by primary intention.

On discharge from the ward, the patient was symptom-free and had gained 6 lb. in weight. A letter from the patient's doctor on Aug. 26 states that he is in particularly good health, is in constant employment, can eat anything and everything without trouble, and is symptom-free.

COMMENTARY

This anomaly of a gastric pouch connected to the main pancreatic duct appears to be unique. I have found no other exactly similar case in the literature.

The problem of nomenclature arises, and although it can be termed a 'gastric reduplication' in that a small second stomach is present, it does not fall into the definition of duplication given by Ladd and Gross (1940). These authors have defined duplications of the gastro-intestinal tract as, "Hollow structures which possess a muscular coat, usually of two layers, lined with epithelium similar to that found in some portion of the gastro-intestinal tract. An important finding is the histological fact that the muscular coats of the duplication are intimately adherent to, and at times are microscopically an integral part of the musculature of the alimentary tract." Elsewhere Gross (1953) stresses this close union, stating that "as a general rule it is quite difficult or impossible to separate one from the other without injuring the bowel".

Of 28 cases of duplication in the region of the stomach and duodenum that have been described (Ladd and Gross, 1940; Ferraro, 1942; Orgias, 1943; Donovan and Santulli, 1947; McCutchen, 1949; Miller and Ginsberg, 1950; Berg and Marx, 1952; Lyall, 1952; Gross, 1953; Ellis, 1953; Owen, Holman, and Priestley, 1954) 26 were intimately united with the normal foregut, and only 2 communicated directly with it.

As the anomaly described was a discrete tumour lying apart from the duodenum, and communicated with it only indirectly via the pancreatic duct, I do not believe it can be regarded as a duplication.

Some of the views put forward to account for diverticula and other similar anomalies follow: Evans (1929) in his Hunterian lecture on "Developmental Enterogenous Cysts and Diverticula", also Ladd and Gross (1940), favoured the view put forward by Lewis and Thyng (1907), who found diverticula in the fœtal alimentary tract of man, pigs, and other mammals. Normally these diverticula, which in all the specimens were found most frequently in the terminal ileum regress, but, should this not occur, one may become pinched off to form a duplication.

In order to adapt this view to the present case, in which a duct from the gastric pouch drains into the main pancreatic duct, it is necessary to regard the pouch as a diverticulum of the latter duct. The presence of such diverticula has not been demonstrated.

Bremer (1944) divided duplications into spherical and tubular types. He stated that the spherical types are derived from diverticula projecting from the antimesenteric border of the alimentary tube, which normally regress but may persist and grow. Some spherical and most tubular duplications arise from abnormal persistence of vacuoles which are present in the 'solid stage' of development. These vacuoles run together to form a channel alongside the normal gut.

This explanation could not account for the present anomaly as it is neither on the antimesenteric border of the gut, nor does it form a channel alongside the normal intestine.

Recently the association between intrathoracic and/or intra-abdominal enterogenous cysts, together with anomalies of the cervical or thoracic vertebræ, has been noted. The hypothesis has been put forward by Veeneklaas (1952), who described such a case,

"that adherence of the notochord and foregut persists. Descent of the stomach would then cause anomalies in the vertebral bodies in this part of the spine while the corresponding part of the stomach would be pinched off."

McLetchie, Purves, and Saunders (1954) reported a case of a separate accessory stomach communicating with the first part of the duodenum. The stomach was partly intrathoracic and there were dense fibrous adhesions between the posterior aspect of the gastric pouch blending it firmly to the vertebræ. The vertebral defect was a cleft of the sixth cervical body. He puts forward an explanation, similar to that of the previous author, stressing the importance of the neurenteric band which may form and cause traction diverticula at one or both points of attachment (neural or alimentary), or give rise to intrathoracic or intra-abdominal cysts. Thus "it would appear that many so-called enterogenic diverticula, cysts, or strands owe their existence to coincident malformation of the embryonic spinal axis which may or may not later express itself as a frank body cleft, or some minor degree of combined spina bifida".

Further cases in the same category were reported by Poncher and Milles (1933), Davis and Salkin (1947), and Gross, Neuhauser, and Longino (1950). Recently Roberts and Weeks (1957) have added 2 more cases to the literature. A feature of all these cases has been the coexistence of a vertebral abnormality together with a fibrous band or adhesions between the cyst and the spine. In the present anomaly these two features are absent.

It is suggested that the present anomaly is an example of heteromorphism in the pancreas. It is postulated that during the development of the pancreas a group of cells, instead of growing into normal pancreatic tissue, underwent heteromorphosis and developed into a small facsimile of the stomach whilst still retaining their connexion with the main pancreatic duct. The main support for this hypothesis comes from Nicholson (1923). Discussing the genesis of an oval pancreatic cyst containing gastric mucosa and pancreatic tissue, from a female patient aged 49 years, he wrote as follows: "I cannot believe that the pancreatic bud in the embryo has any more the 'intention' of giving rise to pancreatic tissue than the rudiment of the hepatic duct has of growing to form the liver. Its cells possess all the potencies of those of the gut of which they have barely ceased to form a part. But in the very process of budding their environment alters. They are thus forced to develop certain of their original potencies and to suppress others. By so doing the immediate environment—the stimuli, internal as well as external, to which these cells are subjected, is again altered. If the sequence of stimuli or changes of environment is orderly the cells will, in the fullness of time, acquire the structural characters of pancreatic epithelium. If it be interrupted and altered to an extent to which the cells can only respond by the development of some other of their original potencies, an abnormal or heterotopic tissue must result." In other words this abnormality represents an unusual differentiation of pancreatic cells resulting from abnormal conditions occurring during development. Nicholson was not prepared to discuss the nature of the abnormal condition which may have affected that part of the pancreas. However, he stated: "It is tempting to speculate upon the possible after-effects of the occlusion of one of its branches, with dilatation behind it. But it is better not to do so, since pathology, in common with all the biological sciences, has suffered overmuch from premature and presumptuous attempts at finding explanations. All that can be done is to attempt to show that an epigenetic origin for this and for similar malformations is worthy of consideration."

SUMMARY

1. An unusual anomaly of foregut development occurring in a young male patient is described.

2. The possible genesis of foregut abnormalities is described in brief.

3. It is concluded that the present anomaly arose by heteromorphosis during the development of the pancreas, and thus should be called a pancreaticogenic gastric diverticulum.

I am grateful to Professor H. Scarborough and to Mr. D. B. E. Foster for permission to publish the case history, and for their interest in this paper. I am greatly indebted to Professor Lambert Rogers for his constructive criticism. The illustrations are by Miss June Williams and the photographs by Mr. Ralph Marshall.

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THE USE OF ROTATION FLAPS FOLLOWING EXCISION OF LUMBAR MYELO-MENINGOCELES*

AN AID TO THE CLOSURE OF LARGE DEFECTS

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THE surgical treatment of myelo-meningoceles is a controversial subject and it is not proposed in this short paper to discuss the indications for operation or the time at which it should be carried out. Once it has been decided that the lesion should be explored and the sac excised, it is essential to achieve complete

FIG. 695.—The circular defect following excision of a large myelo-meningocele can be resolved into two triangles.

closure of the defect with a covering of healthy skin and subcutaneous tissue. In a proportion of cases the defect is too large to allow simple approximation of the skin edges, and rotation flaps from adjacent areas of the back and buttocks have proved useful.

In this paper the factors which may make skin closure difficult are described so that an attempt may be made to assess these factors pre-operatively. If it seems likely that skin closure will be difficult it is an advantage for the neurosurgeon and the plastic surgeon to work together. The cutting of suitable skin-flaps is a standard procedure in plastic surgery, and the method is described here in the hope that it will prove useful to those surgeons who may not be familiar with such methods of closing large skin defects.

Case Material.—*Table I* shows the total number of children with myelo-meningoceles at all spinal

Table I.—TOTAL NUMBER OF MYELO-MENINGOCELES AT ALL SPINAL LEVELS SEEN AT THE HOSPITAL FOR SICK CHILDREN IN SIX YEARS ENDING 1956

	Meningocele	*Myelo-meningocele*	*Total*
Cases seen	105	141	246
Operated on	63	67	130

levels operated upon by all surgeons at the Hospital for Sick Children in a period of six years. On going through the case records it is clear that there was rarely any difficulty in closing cervical and dorsal myelo-meningoceles. When the lumbar myelo-meningoceles only were studied, it was found that 25 per cent of those operated on over a three-year period could not be closed by direct suture (*Table II*).

* The term 'myelo-meningocele' is used to include meningoceles as well as true myelo-meningoceles.

Pre-operative Assessment.—The size of the sac in any given case is no guide to the ease of closure

Table II.—LUMBAR MYELO-MENINGOCELES OPERATED ON IN THREE YEARS ENDING 1957

Total operated on	—	56
Closed by direct suture	42	—
Closed with relieving incision	2	—
Closed with skin-flaps (in 7 of these the flaps were cut as described in this paper)	12	—

following excision, and, in fact, one-third of the cases treated by direct suture had sacs which were very large.

Following excision of a large sac, the stretched skin at the margin retracts and thickens, but rarely proves strong enough for a permanent repair by direct suture. This marginal skin may also be involved in a hæmangiomatous malformation which makes it unsuitable to hold the strong sutures which may be required. Once the fringe of retracted abnormal skin has been excised around the margin the surgeon may be faced with an unexpectedly large defect.

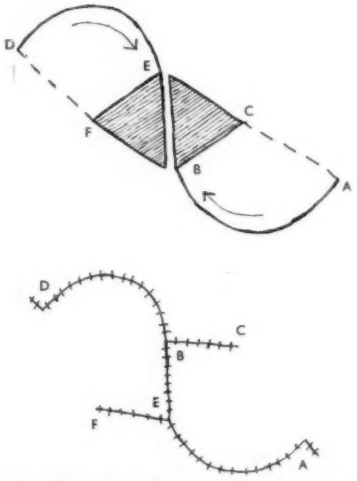

FIG. 696.—These triangular defects can be closed by rotation flaps of skin and subcutaneous tissue. The width of the bases AC and DF should be equal to the length of the flaps.

The most important factor in the pre-operative assessment is the size of the neck of the sac; a sessile type of sac is always more difficult to close than the more common sac with a narrower neck. In practice we have found that when the diameter of the neck of

the sac is more than half the width of the back, watertight closure of the defect will not be possible by simple approximation.

We have, therefore, assessed our cases before operation with particular attention to these two facts: (1) The size of the neck of the sac in relation to the width of the back; (2) The condition of the skin at the neck of the sac.

FIG. 697.—Myelo-meningocele before excision. All thin and discoloured skin is trimmed away from the neck of the sac.

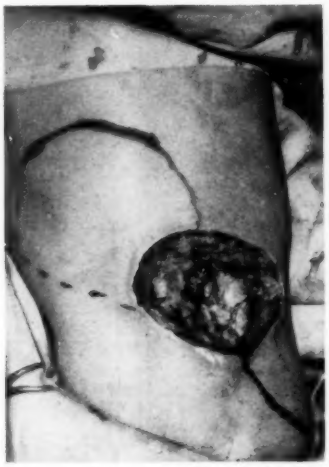

FIG. 698.—Defect left after excision of the sac. Two rotation flaps marked out.

FIG. 699.—Flaps raised and rotated into position without tension.

FIG. 700.—Suturing started at the centre.

FIG. 701.—Suturing completed.

Pre-operative Preparation.—Since this short article is concerned only with a particular method of closing large defects, there is but one point about pre-operative preparation which needs special mention. The area of skin involved in the rotation flaps is large, so that the skin of the flanks, back, and buttocks must be free from any septic lesions before operation is undertaken. Similarly, the covering of the myelo-meningocele should be thoroughly cleaned. It is frequently necessary in older cases to remove desquamated skin and debris by sponging with an antiseptic detergent such as Cetavlon for several days before operation.

Technique of Closure by Rotation Flaps.—The procedure which has been found most effective is that known to plastic surgeons as the 'S rotation flap'. It is frequently used in the closure of bedsores and defects in the lumbar and sacral regions.

All rotation flaps depend for their planning on the resolution of the defect into a triangle or triangles.

The circular defect that results from excision of a meningocele can be considered as two triangles (*Fig.* 695), which can be closed by two flaps rotating in opposite directions (*Fig.* 696).

Following removal of the sac, all thin and discoloured skin is trimmed away (*Fig.* 697). The flaps are marked out in ink; each flap is so planned that the width of the base is equal to the length of the flap (*Fig.* 698). The skin incisions are made with a knife and the flaps are raised in full thickness down to the deep fascia with diathermy. There is the danger in using diathermy on any skin-flap of producing a spreading thrombosis of vessels which are essential to the nutrition of the flap. In these myelo-meningoceles the risks are outweighed by the advantages of saving of time and blood loss in what may be a severe operation in a small baby. Care must be taken to make the incision in the skin with a knife and to keep the diathermy point away from the skin edge.

When the flaps are raised in their full extent and rotated into their new positions they should lie without tension (*Fig.* 699). Suturing in one layer is started at the junction between the flaps (*Fig.* 700). Once this central area is safely covered, suturing is continued back towards the base. The sutures are inserted to advance the flap along the fixed side and so ensure that there is no tension on the centre (*Fig.* 701). There is thus raised up a 'dog-ear' of excess tissue on the fixed side near the base of the flap; this can be trimmed off away from the flap so that the base of the flap is not narrowed. This is a convenient site for the insertion of a drain which is important in preventing the accumulation of fluid under the flap.

Post-operative.—The child is nursed post-operatively in the prone position, the dressings being carefully arranged to avoid pressure on the bases of the flaps. As is usual in all operations upon lumbar myelo-meningoceles, the foot of the bed is raised to reduce the cerebrospinal fluid pressure in the region of the dural repair.

SUMMARY

After excision of lumbar myelo-meningoceles, closure by direct suture of the skin is not possible in about 25 per cent of cases.

The factors which may indicate pre-operatively that a plastic procedure will be necessary are mentioned.

A method of closing large defects using rotation flaps is described.

Acknowledgements.—We would like to thank Mr. D. N. Matthews and Mr. Wyllie McKissock for help in the preparation of this paper and for permission to treat some of these patients.

We are grateful to the Medical Committee of the Hospital for Sick Children for permission to study these case records and to publish the results.

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THE PLACE OF SURGERY IN THE MANAGEMENT OF INTRACTABLE PAIN

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PAIN as an indication of injury by trauma or disease leading to appropriate treatment is a sensation of great value to the individual. However, when its cause is irremovable, long-continued and severe pain is entirely harmful and productive of severe psychological and physical change in a patient, a state of affairs likely to be worsened by the development of addiction to the narcotic drugs which alone provide a measure of relief. For centuries the unhappy state of such patients presented an unanswerable challenge to the physician. Accounts of early attempts to relieve them by surgery describe such measures as stretching or section of major peripheral nerves, amputation, and section of the spinal cord, and indicate not only the desperation which the situation aroused but the meagre benefits resulting from the methods available.

As knowledge of the anatomy and physiology of the central nervous system developed, the possibility of relieving intractable pain by more appropriate section of nerve-fibres attracted attention. Thus Bennet in 1889 first reported interruption of the pain pathway by division of the posterior spinal roots and in 1912 Spiller and Martin described its interruption by section of the spinothalamic tracts. In the evolution of the treatment of facial pain the work of Horsley (Horsley, Taylor, and Coleman, 1891) and Rose (1902) in this country, and that of Hartley (1892) in the United States and Krause (1893) in Germany, on excision of the Gasserian ganglion led in due course to the modern operation of division of the sensory root of the ganglion first described by Spiller and Frazier in 1901. The interruption of the trigeminal pain pathway by injection of alcohol into the ganglion or peripheral divisions of the nerve first described in 1906 was a further contribution. Subsequently intracranial section of the glossopharyngeal nerve was described (Dandy, 1927), and later the section of pain pathways within the brain-stem; the first of the latter operations, described by Sjoqvist in 1938, being section of the descending trigeminal tract in the medulla oblongata. Finally in 1946 it was shown by Freeman and Watts that the operation of pre-frontal leucotomy, while leaving a patient's pain pathways and perception of pain intact, could so modify his reaction to the sensation as greatly to relieve his suffering.

Although a considerable number of surgical procedures directed at various levels in the nervous system and designed to relieve intractable pain are now available, their value as a whole is not established and their individual merits and demerits are not clearly

defined. The introduction of new forms of surgical treatment can lead to optimistic claims which must later be revised; increased experience may show that the beneficial effects of the procedures are not enduring and that undesired sequelæ occur with a frequency not at first suspected. Reviews of the results of the various pain-relieving operations are clearly necessary.

Table I.—Operations for Intractable Pain in 100 Patients

1. *Rhizotomy*:—		
Trigeminal sensory root alone	5	
Trigeminal sensory root combined with glossopharyngeal and/or cervical posterior roots	4	
Spinal posterior roots alone	10	
		19
2. *Prefrontal Leucotomy*		5
3. *Anterolateral Chordotomy*		80
	Total	104

While analyses of these results from the anatomical and physiological viewpoints have obvious value, it is of first importance to consider them from the viewpoint of the patient and his family; the degree of relief, its duration, the risks to life and function, and the added suffering occasioned, all demand careful consideration. Although groups of cases treated surgically for the relief of intractable pain have now been reviewed by several surgeons, largely in the United States, no such review by a British worker has been encountered. Further, even in large series of cases such as those of Grant (1943) and White and Sweet (1955) no adequate indication of the duration of pain relief after operation is provided. This is a serious omission, since the value of an operation designed to relieve intractable pain is not established by the demonstration of analgesia in the desired area a few days or weeks after the procedure; nor indeed is there real value in the claim that pain has been relieved unless the duration and permanence of the relief are indicated.

In this paper the records of 100 patients suffering from intractable pain and treated by various operations upon the central nervous system are reviewed. It is hoped that the findings may help to define the place of surgery in the management of such cases by indicating the measure of relief obtained in the patients of the series as well as its cost in terms of mortality and undesirable sequelæ. The patients whose records have been reviewed form a consecutive personal series dealt with over a 16-year period to October, 1957, almost entirely in the Department of Neurological Surgery of St. Bartholomew's Hospital. In 87 patients the pain was occasioned by malignant disease and in 13 was due to other causes. Many more operations have been performed for the relief of trigeminal neuralgia than for all other causes together, but this group is excluded from consideration, as also are operations upon the sympathetic nervous system for the relief of visceral pain and causalgia. In the 100 patients 104 operations were performed; one of the trigeminal rhizotomies had a subsequent prefrontal leucotomy, and a spinal posterior rhizotomy was followed by an anterolateral chordotomy in another case; in 2 patients a second anterolateral chordotomy was carried out. The types of operation and the number of cases in which each was performed are indicated in *Table I.*

SENSORY RHIZOTOMY

The rhizotomies in the series numbered 19 and can be divided into three groups: (1) Trigeminal sensory root section alone; (2) Trigeminal rhizotomy combined with section of the upper cervical posterior roots and sometimes the glossopharyngeal nerve; and (3) Section of spinal posterior roots. These groups will now be analysed separately.

1. Trigeminal Sensory Root Section.—Trigeminal sensory root section alone was performed in 5 cases, the approach being the classical middle fossa one of Frazier in 4 cases and that of Dandy through the posterior fossa in the remaining 1 case. The ages of the patients varied from 21 to 68 years; 4 were males and 1 a female. The root section was total in 3 cases and fractional in 2. The lesions responsible for the pain were a sarcoma of the maxilla, and the carcinomata of the maxillary antrum, the floor of the mouth, and the parotid gland; the remaining patient had cranial metastases from a primary breast carcinoma. In 2 patients complete relief of pain till death after 30 weeks and until lost to follow-up at 4 weeks was obtained. In the other 3 pain re-appeared, though not in the denervated area: in one after 25 weeks and as a result of widespread infiltration of the skull base by an antral carcinoma which occasioned pain in the trigeminal territory of the opposite side, as well as in that of the spared ophthalmic fibres on the side of the fractionally sectioned root; the patient with metastases in the cranial base from a mammary primary developed pain in the opposite trigeminal territory, and the one with a parotid carcinoma developed otitic pain, both 4 weeks after operation. There was no mortality in the group, but two undesired sequelæ occurred: one patient developed a facial paralysis after operation, and another, whose skull base was widely infiltrated by a parotid growth, had a transient cerebrospinal fluid otorrhœa. The procedure entailed but slight post-operative suffering.

Considering this small group of trigeminal rhizotomies in retrospect, the recurrence of pain was due to the malignant disease spreading to involve both sides of the skull base in 2, and the advance of the disease process rather than inadequacy of the surgery must be regarded as the cause of failure. In the third case, since the middle ear was infiltrated, an adequate denervation would have necessitated section of the ninth cranial nerve and upper cervical posterior roots, an operation for which the patient was considered to be unfit.

Thus trigeminal rhizotomy has but a small place in treatment of such cases, since frequently the infiltrating neoplasm will involve pain fibres in other nerves on the same or opposite side of the head. Where the pain-producing lesion is not so situated as to prevent it, alcohol injection of the Gasserian ganglion is an alternative procedure and had been previously tried elsewhere in certain of the cases of the series without success.

2. Extended Trigeminal Rhizotomy.—In 4 cases trigeminal rhizotomy was combined with section of the upper two or three cervical roots on the affected side, with the addition of the glossopharyngeal nerve in 3 of them. Three of the group were male and 1 female, their ages varying from 31 to 67 years. In the 3 patients in whom the rhizotomy included

the glossopharyngeal nerve pain resulted from carcinomata of the tonsil, floor of the mouth, and parotid gland, and in the patient in whom this nerve was spared an epithelioma of the face: all had metastases in cervical lymph-nodes. All of these patients obtained complete relief until they died or were lost to follow-up; the period of relief being in individual cases 4 weeks, 3 months, 7 months, and 11 months. There was no mortality nor any immediate post-operative complication, although the patient with the parotid carcinoma developed an infection of the meninges—thought to arise in the middle ear—five weeks after operation. This led to a dorsal sub-arachnoid abscess and a paraplegia which was relieved by laminectomy and evacuation of the abscess. The more extensive exposure in these cases might be expected to occasion greater post-operative pain than in the previous group, but this has not been severe or persistent.

When this group is contrasted with the previous one it is clear that the wider sensory denervation gives rise to a more certain and lasting relief, and if a tentative conclusion is justified it is that in cases of the type under consideration it is to be preferred to simple trigeminal root section.

3. Section of the Posterior Spinal Roots.—In 10 cases section of the posterior spinal roots was performed. The patients' ages varied from 43 to 79 years, 7 being male and 3 female. Five of them had bronchial carcinoma involving the chest wall, 1 a paravertebral carcinoma possibly of pancreatic origin, and 1 a lumbar vertebral metastasis from a thyroid carcinoma; in 3 the pain was secondary to a non-malignant condition—a thoracotomy scar in one, a vertebral compression fracture in another, and in the final patient the cause of the pain remained undiscovered. In 2 patients both the pain and the rhizotomy were bilateral and the number of roots sectioned varied from two to six. In 5 patients, 3 with malignant disease, pain was completely relieved—in the cases with carcinoma for an average survival period of 5 months; in 3 patients, all with carcinomata, pain re-appeared after 2, 4, and 5 weeks; in 2 of these pain developed outside the analgesic area and in the third recurred at its previous site within this area. In 1 patient with posterior thoracic pain of unknown causation, section of five posterior roots produced analgesia in the desired area but pain was quite unaffected. This man was addicted to pethidine and after his death 2 months post-operatively no cause for his pain was revealed. One patient with a bronchial carcinoma in whom surgical treatment of pain had been delayed for twelve months died during the operation. In the surviving patients there were no undesirable sequelæ and pain in the laminectomy wound was not a troublesome post-operative symptom.

In considering these cases of posterior rhizotomy certain points emerge. First the futility of a late operation upon a patient so exhausted not only by the progression of his disease, but by months of severe pain that narcotic drugs in adequate dosage for a few weeks is all that is required; secondly, the desirability of determining the cause of an intractable pain, especially where the possibility of drug addiction exists, before attempting surgical treatment; thirdly, the absence of unwanted sequelæ in the surviving patients is noteworthy; finally, of the 8 patients in whom the proper indications for operation were present, 5 were completely relieved and 2 developed pain outside the analgesic area from progression of the disease. In only 1 case did pain recur in the denervated territory.

PREFRONTAL LEUCOTOMY

Prefrontal leucotomy was performed in 5 patients whose ages ranged from 37 to 72 years. Two of these had carcinomata of the maxillary antrum; in one an infected neoplasm had ulcerated on to the cheek and in the other recurrence had developed in the skull base following a radical removal fourteen years previously; of the remaining 3 patients one had a carcinoma of the tympanum, another a recurrent carcinoma of the floor of the mouth, and the last recurrence of a renal carcinoma. All suffered severe, continuous pain, and the possibility of an operative procedure to denervate the lesion was carefully weighed but was considered to be contra-indicated by the patient's age in 1 case, cachexia in 2 cases, and the encroachment of the malignant ulcer on what would have been the operative approach in 1 patient; 1 of the 5 cases had a previous trigeminal root section with relief for 6 months. After operation pain was no longer a complaint in two patients and was so greatly reduced in another that it was possible to discontinue the administration of morphine and heroin; in 1 patient pain was reduced but was still troublesome when food came into contact with the malignant ulcer in his mouth. The fifth patient remained in a stuporose state until his death on the twelfth post-operative day. Excluding this operative death the period of survival of the remaining 4 patients was 32, 63, 120, and 150 days after operation, and during this period tension and apprehension remained greatly reduced or was absent. Undesirable sequelæ were present in all cases: 1 remained withdrawn and incontinent during the five months of her survival, and 3 showed a marked loss of insight and reduction of affect.

The fact that leucotomy is technically a simple procedure, and that it will be tolerated even by extremely ill, cachectic patients, would suggest that it provides a satisfactory answer to the intractable pain of late malignant disease. However, on reflection it is clear that an operation which will remove the tension and apprehension associated with the pain of incurable disease will do so only at the cost of a major change in personality. The loss of emotional response and insight is not limited to the patient's pain and the disease causing it, but is reflected through the whole range of emotional and intellectual activity. The pain remains, but the personality is so altered that the sensation ceases to carry any unpleasant significance. While the patient is doubtless unaware of the change it can be extremely distressing to his family who suffer a double deprivation—the loss of the known and loved personality being followed after an interval by death in the usual sense of the word. This form of therapy must clearly be avoided whenever possible and instituted only as a last resort after full explanation of its implications to the family. In patients whose misery stems from a number of causes —severe pain, an ulcerating and enlarging growth, the realization of the inevitability of continued

deterioration—and whose morale is all but broken, the procedure can bring relief to all concerned.

ANTEROLATERAL CHORDOTOMY

Anterolateral chordotomy designed to section the spinothalamic pain pathway has been performed in 78 patients. One of these had had a previous posterior rhizotomy without lasting benefit, and in 2 cases a second chordotomy was performed after intervals of 11 months and almost 4 years. The ages of the patients in this group varied from 6 to 75 years; 5 patients were under the age of 30 years and 13 over 60 years, 40 being in the fifth or sixth decades of life. In 15 cases the section was affected in the upper cervical cord, bilaterally in 1 case, the usual indication for this high level of section being pain in an upper limb; in 63 cases the cord section was carried out in the upper dorsal region at about the level of the fifth dorsal segment, the section being bilateral in all except 1 case. In 10 patients the chordotomy was performed for pain due to causes other than a neoplasm, e.g., a painful phantom limb or pain secondary to a spinal injury; in 68 patients it resulted from inoperable malignant disease—carcinoma of the cervix and rectum and sarcomata of various types accounting for two-thirds of the cases. Of the 78 patients, 6 died within 4 weeks of operation, 2 within 1 week. This matter will be referred to in the general discussion to follow, since these operations might have been avoided by a more careful process of selection.

Table II.—Relief from Pain following Chordotomy in 72 Cases

Complete and permanent relief		45
Incomplete relief:—		
Recurrence or development of pain outside area of analgesia after initial complete relief	16	
Partial relief	6	
		22
No relief		5
Total		72

The value of anterolateral chordotomy in relieving pain in the 72 patients surviving operation by more than 4 weeks is summarized in *Table II*. In the 45 patients completely relieved of pain there was no recurrence until the patient died or was last heard from, the period ranging from 4 weeks to 7 years. The period of known relief was less than 6 weeks in 13 patients; 7 to 12 weeks in 11 patients; 13 to 26 weeks in 6 patients; and over 26 weeks in 15 patients. In 16 of the 72 patients initial complete relief was followed by recurrence of pain to a greater or lesser extent; in 13 of these pain recurred in part of its previous area of distribution and in the remaining 3 it appeared in new territories outside the analgesic area. As regards the former group it is noteworthy first that in half of them the pain which re-appeared after chordotomy was considered by those caring for the patient to be of reduced severity and remained so until death; and, secondly, that in certainly 10 of the 13 patients a fading of post-operative analgesia to hypo-algesia, especially in the sacral dermatomes on one or both sides, occurred in association with the return of pain. As regards the 3 patients who developed pain outside the area of analgesia, their original pain was relieved and the extension to new territories was due to spread of the disease. A patient with a painful phantom upper limb was relieved of the pain for 3 months, then developed cervical and thoracic pain, with evidence of sarcomatous metastases. Another with crural pain secondary to involvement of the sacral plexus by spread of a carcinoma of the cervix was relieved for 4 months and then developed anterior and posterior abdominal pain above the level of the analgesia (D.10). The third patient with a lower dorsal paravertebral growth of possible pancreatic origin was relieved of abdominal pain but developed pain above the analgesic level (D.7) in the chest and shoulders from involvement of the diaphragmatic nerve-supply.

In 6 of the 72 patients operation relieved part of the pain only. In 1 patient with a sarcoma of the pelvic wall a satisfactory analgesia was achieved with cure of lumbar and crural pain, but a rectal ache persisted; in the remaining 5 patients pain recurred in areas in which sensibility re-appeared, but permanent relief was obtained in the greater part of its previous distribution; in 2 of them pain and paræsthesiæ developed in the sacral dermatomes; in another 2 pain persisted in a limb in which operation had produced hypo-algesia only, and in the remaining 1 case a fall in the sensory level caused a recurrence of abdominal pain although crural pain was relieved. These patients were, for the most part, benefited by operation—in 3 during known periods of 16, 30, and 100 weeks.

In *Table III* the results of anterolateral chordotomy in a group of 58 patients relieved of pain completely either permanently or with subsequent recurrence are analysed after post-operative periods of 6, 12, and 24 months; the 3 most recently treated patients in the total group of 61 such patients are excluded. It will be seen that of the patients alive at each of the stated intervals after operation almost three-quarters were completely relieved of pain. Of the patients who were dead, more than half had died within 6 months of operation, and the proportion completely relieved at death was but slightly lower than in the group of surviving patients. The third group of patients presumed dead were not followed up until death, which was thought to have occurred within a few months; each of these 9 patients was known to have been relieved at the time of discharge from hospital.

Table III.—Analysis of Results of Chordotomy in 58 Patients at the Stated Post-operative Periods

		Position at Following Periods Post-operatively		
		6 months	1 yr.	2 yr.
Alive	With relief	19	12	7
	Without relief	6	3	2
Dead	With relief until death	18	24	28
	Without relief at death	6	10	12
Presumed dead	With relief for at least part of time	9	9	9
	Total	58	58	58

The value of chordotomy can also be shown by calculating the total number of completed months in

the first 2 years after operation and the number of months of complete relief obtained. Adequate information for such a calculation was available in 52 of the patients; the remainder of the 72 patients surviving operation are excluded from the calculation because the operation was too recent to permit inclusion (2 cases) or because the follow-up was not completed (7 cases), or because complete relief of pain did not persist for a full month post-operatively (11 cases). The total number of complete months survived in the first two years after operation in these 54 patients was 510 out of a possible maximum of 1248 months (i.e., 41·5 per cent). In these patients there was complete relief in 361 of the months survived—that is 70 per cent.

In 5 of the 72 patients surviving chordotomy pain was entirely unrelieved. In 2 of these it is thought to have been psychogenic and chordotomy should not have been performed; in the remaining 3 patients there was no relief either because the incisions in the cord produced no adequate sensory loss or because the level chosen for the cord section was too low.

To summarize, of the 72 patients surviving chordotomy for more than 4 weeks, 45 (62·5 per cent) were completely relieved; 22 (30·5 per cent) were incompletely relieved in that pain either recurred, developed in new territories, or was reduced but not abolished; and 5 patients (7 per cent) were completely unrelieved.

Complications of Chordotomy.—Following section of pain pathways within the spinal cord undesired sequelæ are frequent, and include disturbances of sphincter control, a motor defect, and pressure sores. Such sequelæ may occur in spite of the most careful operative technique, a fact which indicates that the spinothalamic tract is neither so circumscribed nor so separated from neighbouring tracts as to render possible its complete section with preservation of all neighbouring fibres of different function.

1. Sphincter Disturbance.—As regards the anal sphincter this may be noted to be hypotonic, and in the deteriorating cachectic patient rectal incontinence may occur, especially when stools are loose. As is true of spinal cord injuries in general, rectal incontinence is, however, rarely a serious problem. In the case of the vesical sphincters the position is entirely different. In the 13 patients surviving cervical chordotomy disturbance of micturition was always quite transient; this is doubtless to be associated with the fact that in all except 1 of the patients the cord section was unilateral. Of the 59 patients surviving dorsal chordotomy (bilateral in 58) 2 had had pre-operative suprapubic cystostomies because of a spinal cord injury and must therefore be excluded from consideration of vesical disturbance after chordotomy. In the remaining 57 patients post-operative retention of urine occurred regularly and commonly persisted for 2 to 3 weeks. In 38 of this group of patients voluntary micturition was restored; however, precipitancy of micturition and occasional incontinence (3 cases) were not infrequent, and with progression of the malignant lesion and deterioration in the patient's general condition late incontinence is known to have developed in 5 cases. In 11 cases complete voluntary control was not regained; here injections of carbachol produced a good response and voluntary micturition with a considerable amount of residual urine was present; in addition stress incontinence might occur. In 8 cases loss of voluntary control was complete, with persistent retention in 6 and incontinence in 2. These disturbances persisted for from 3 to 12 weeks, until the patient's death or last reporting. In 2 of the group of 8 patients an abdominal mass prevented the bladder from filling and in the remaining 6 there was severe cachexia. Thus if the unilateral cervical cases (12) and those with cystostomies (2) are excluded from the 72 surviving patients, all of the remaining 58 patients showed disturbance of vesical control. In two-thirds (39 cases) this was transitory, although it might re-appear as cachexia developed. In one-third of the group (19 cases) the disturbance was persistent until the patient died or was lost to follow-up—a period of 3 to 12 weeks from the time of operation; in rather more than half of the 19 cases (11 cases) voluntary micturition was present, but chronic retention necessitated carbachol injections and catheterization at intervals; in the remainder (8 cases) the loss of control was complete. Advancing age, increasing weakness, and progressive infiltration of the bladder itself or its nerve-supply all contributed to the disturbance of vesical control, yet it remains clear that section of the anterolateral columns of the cord interferes with the innervation of the urinary bladder. While the effects of this may be transient they are not always so, and if they do pass off they may recur. The risk of urinary infection and its unfavourable systemic effect upon a debilitated patient make even temporary urinary retention highly undesirable.

2. Loss of Voluntary Power.—Section of the anterolateral column also carries a risk of impairment of voluntary power from injury to descending motor pathways. In 10 (14 per cent) of the 72 surviving cases this complication occurred. In 3 cases there was a paralysis or severe paresis of a lower limb, and in each of these some degree of reduction of power persisted; in 7 cases the weakness took the form of a mild paresis which cleared completely in 4 but was persistent in 3. Theoretically the corticospinal fibres lying posterior to the attachment of the dentate ligament should escape injury from a correctly placed incision in the anterolateral column of the spinal cord. However, in the presence of an ill-defined dentate ligament, or where bleeding occurs from the incision, the upper motor neurones can be damaged.

3. Pressure Sores.—The third important complication of chordotomy is the development of pressure sores. These occurred commonly in a minor form, but in 8 (13 per cent) of the 58 surviving patients in whom a bilateral dorsal chordotomy had been performed large pressure sores developed in the trochanteric or sacral regions and contributed to the rapid deterioration of the patient's general condition. This complication is due to the combination of analgesia of the pressure areas, malnutrition and general asthenia from the prolonged pain, the inevitable use of narcotics, and frequently the presence of malignant disease. Only by attention to nutrition and the most meticulous nursing care can their incidence be reduced, and it must be remembered

that the risk of such a complication will continue after the patient leaves hospital.

4. **Post-operative Pain.**—After chordotomy pain at the site of operation, which in the case of upper dorsal chordotomies radiates as a girdle to the upper anterior chest, is a frequent and sometimes troublesome symptom and has been commented upon by numerous authors. In cervical chordotomies section of the upper cervical posterior roots renders the area of the wound analgesic and greatly diminishes discomfort. A similar rhizotomy in the case of the dorsal operation does not affect sensibility in the area of the wound; neither does it reduce the girdle pain, which is thus shown not to be the result of injury to roots at the time of operation. The exact cause of the symptom is not clear, though it seems possible that pain at the site of the operative incision is experienced more vividly on a background of complete analgesia in areas distal to wound level than is the case when sensibility in these areas is undiminished as it is after most laminectomies. As Kahn (1955) has emphasized, a careful operative technique will reduce post-operative wound pain. In the present series it has rarely persisted for more than a few weeks.

Thus while the operation of anterolateral chordotomy relieved pain either totally or partially in a high proportion (90 per cent) of the 72 patients surviving operation, it did so at the cost of persistent disturbance of vesical control—of greater or less severity—in 26 per cent, a persisting motor defect in 8 per cent, and pressure sores in 11 per cent.

DISCUSSION

The requisites of an ideal surgical procedure for the relief of intractable pain can be clearly laid down. The operation should provide complete and lasting relief and produce neither a functional defect nor any undesirable sequelæ; further it should be commensurate with the patient's strength and occasion no added suffering in itself. Clearly all the available procedures fall short of this ideal, and indeed were this not so the problem of intractable pain would have been entirely solved by surgery. None of the surgical techniques described ensures certain and permanent relief from pain. Thus if the results in the 94 patients who survived the different types of operation which have been described above are considered together, in 58 (62 per cent) complete relief was obtained, in 22 (23 per cent) relief with recurrence, and in 14 (15 per cent) pain was relieved only in part or not at all.

A functional defect is inevitable after every operation directed at the pain pathways. In cases of rhizotomy the sparing of roots carrying proprioceptive afferents from the limbs will ensure against a motor defect, while the loss of cutaneous sensibility carries but a small risk of injury to the skin and the development of trophic sores. In contrast, the analgesia of the pressure areas produced by chordotomy brings a considerable threat of cutaneous ulceration. Serious pressure sores occurred in 13 per cent of patients surviving a bilateral dorsal chordotomy. Chordotomy also carries the risk of a motor defect—a disturbance which occurred in 14 per cent of the 72 cases surviving operation, there being a persistent defect in 8 per cent. Bilateral chordotomy is commonly followed by disturbance of vesical control; this was transient in 68 per cent but persisted in greater or less degree in 32 per cent. In cases treated by leucotomy there was a marked change in personality in each of the surviving patients.

Operative Mortality.—The inability of the patient to tolerate the operative procedure is in part indicated by the operative mortality. One of the 19 patients submitted to sensory rhizotomy (unilateral spinal posterior root section) died before the termination of the operation; this cachectic man had suffered severe pain for 16 months, and autopsy revealed a suprarenal metastasis. One of the 5 patients treated by leucotomy died on the twelfth post-operative day; this patient was cachectic, and remained drowsy after operation, succumbing to bronchopneumonia. Six of those submitted to anterolateral chordotomy died within 24 days of the operation. Two of these patients developed increasing pulmonary œdema secondary to intrathoracic metastases; of 2 others with phlebothrombosis secondary to carcinomata of the cervix, 1 died from pulmonary emboli and the other with auricular fibrillation and multiple metastases; another patient developed bronchopneumonia and in the sixth the cause of death was not determined. The operative mortality for the whole series is therefore 8 per cent. In none of these cases did death appear to be attributable to some error of technique, and it is probable that in every case the disease had reached a stage when surgical treatment of pain should have been avoided. This is particularly so in the case of chordotomy, where urinary retention is so common and where in cachectic patients the risk of urinary infection and pressure sores with resulting depletion of the patient's small remaining reserves is high. The view has been expressed (Stookey, 1943) that operative mortality-rates are meaningless in connexion with intractable pain. However, anything which adds in any way to the suffering of the patient dying of malignant disease is to be regretted and a useless operation constitutes added suffering.

Finally it is clear that all forms of surgery occasion mental and physical suffering in some degree. When an operation is curative such suffering will be accepted; when it is palliative and when life expectancy is to be measured in weeks rather than years the possible benefits of surgery must be carefully weighed in each case against the added suffering which it may entail.

Indications for Operation.—Surgery is indicated in the management of pain where the cause is not amenable to treatment, when this pain is of such severity and when life expectancy is of such duration as to justify it. The severity of a patient's pain is assessed by the extent of its interference with his activities and sleep, as well as its failure to respond to analgesics other than those producing addiction. Estimation of the likely survival period will be based upon the nature of the causative disease and the history of its evolution as well as upon the patient's age and general nutrition. Surgery should be reserved for cases in which pain is severe and unresponsive to simpler measures. It should be withheld when life expectancy is so short that the suffering occasioned by the operation would persist through the period of survival. This suffering due to operation will to some extent depend upon the type of surgery, since the discomforts of a trigeminal rhizotomy are

considerably less than those of an anterolateral chordotomy. The minimum life expectancy justifying operation has been taken to be 12 weeks, although where pain has been intensely severe a shorter expectancy has been accepted; in the 88 patients of the present series for whom the information is available two-thirds survived for 3 months or more and one-third died in under 3 months. When deterioration is likely to be rapid it is clearly the proper course to resort to the stronger analgesic drugs and to increase the dose of these as indicated. If metastases are widespread and there is pain arising in relation to one of these, operative treatment is probably better avoided in most cases since pain is likely to re-appear within a short period in a new territory.

Contra-indications to Operation.—While the indications for surgery are to some extent relative, there are absolute contra-indications. In the first place the suffering of a patient with late malignant disease may have other causes besides pain. An enlarging tumour, the discharges from an infected ulcer, and fistulæ involving the pelvic viscera may all occasion great distress. So also may the steady deterioration in weight and strength associated with cachexia. Pain may be a minor feature in such cases and interruption of the pain pathways can do nothing to remove the other causes of suffering and may well add to the patient's difficulties.

It is of considerable importance that the patient understand that a rhizotomy or chordotomy will relieve the pain alone and will not affect other aspects of his condition—otherwise he may undergo a valueless operation in his efforts to obtain cure of his disease. Indeed, even when pain is relieved by such an operation the patient may become more aware of aspects of his disease which previously troubled him little. Drug addiction is a contra-indication to surgery in cases of intractable pain. When the patient with malignant disease has developed addiction it would seem kinder to avoid the distress occasioned by curing the addiction and therefore to continue treatment with appropriate analgesic drugs. When the pain is occasioned by some non-malignant condition and the expectation of life consequently considerably greater, drug addiction would not constitute a contra-indication to operative treatment. However, efforts to treat the addiction should be made before operation; in this way unnecessary operations for pain which has no organic basis and which therefore cannot be benefited by surgery will be avoided.

A further and frequently related contra-indication to interruption of the pain pathways is psychogenic pain. Its diagnosis may be difficult, though the absence of an organic lesion to explain the severity of the suffering is one indication. Theoretical considerations and practical experience (reported in the literature) make it abundantly clear that even repeated assaults on the pain pathways leave such patients not only unrelieved, but in a worse state than before. In a very occasional case of this sort prefrontal leucotomy may be justified.

When surgical treatment for the relief of intractable pain is indicated in a particular case the appropriate operation must be carefully selected and the technique of its performance should be such as to minimize additional suffering due to the procedure. As to selection of the appropriate operation in unilateral malignant disease of the mouth or nasal sinuses, trigeminal sensory root section through the temporal approach or destruction of its ganglion by alcohol injection may be adequate. However, when the position of the neoplasm or its extensions is such that the territories supplied by the ninth cranial nerve or the cervical plexus are likely to be early involved, a posterior fossa approach combined with an upper cervical laminectomy is indicated. Section to the roots of the fifth and ninth cranial nerves and the upper three cervical posterior ones can thus be effected, and where the patient's condition justifies this more extensive operation the likelihood of lasting relief is considerably increased. Pain in the trunk may be cured with the smallest risk of unwanted sequelæ by a well-planned posterior rhizotomy. When involvement of the limbs is present, as is frequently the case in the common pelvic malignant tumours, chordotomy—in spite of the risks of neurological defects and the greater difficulty in obtaining a lasting analgesia—must be performed, since posterior rhizotomy would produce a useless limb. Finally, prefrontal leucotomy should be reserved for the occasional patient in whom the indications already discussed are present.

Operative Technique.—As to the technique employed, the object should be to minimize further suffering. General anæsthesia should be employed since it seems unjustifiable to submit a patient worn out by severe pain to a prolonged operation under local analgesia and furthermore to ask for his co-operation in sensory tests. While local anæsthesia was sometimes utilized in this series it has been abandoned in the belief that such operations are an ordeal for the patient and in the realization that sensory data obtained may be misleading, since an analgesia at the time of operation may fade to a hypo-analgesia after a longer or shorter interval. It is clearly also desirable to ensure that the patient be spared the ordeal of repeated operations and it is for this reason that such operations as trigeminal tractotomy, which are less certain in their results, have not been employed in the present series. A careful operative technique will reduce pain due to the procedure. In the case of chordotomy in the cervical region posterior rhizotomy will minimize pain in the wound by producing regional analgesia; in dorsal chordotomy pain in the operation site can be reduced by limiting the extent of the laminectomy to a couple of neural arches. Good post-operative nursing will further aid in minimizing suffering.

From the consideration of these cases it is believed that surgery is of value in the treatment of intractable pain. In selected patients when the most appropriate operation is well chosen, carefully timed, and efficiently performed, a very worthwhile relief may be achieved. Since in many of these patients the expectation of life is at best but a few months, every care must be taken to reduce suffering due to the operation—the balance between success and failure in individual cases being frequently a fine one.

SUMMARY

1. The results of surgery in 100 patients suffering from intractable pain are analysed.

2. The advantages and disadvantages of the operations performed in the series—sensory rhizotomy, anterolateral chordotomy, and prefrontal leucotomy—are described.

3. The indications for and contra-indications to surgery in cases of intractable pain are discussed.

In the care of the patients in whom the observations in this paper are based I am indebted to the Nursing Staff of the Hospital and to my house surgeons and chief assistants during the period under consideration. In the analysis of the results of the operations the advice of Mr. M. P. Curwen, the Hospital Statistician, has been valuable.

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CHRONIC LOCALIZED CONSTRICTIVE PERICARDITIS

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INFECTION of the pericardium is probably followed in the majority of cases by resolution, but it may lead to:—

1. Complete or partial obliteration of the pericardial sac.

2. Thickening and contracture of all or part of the pericardium.

3. Pericardial adhesions to the surrounding structures.

The first group of partial or complete obliteration of the pericardial sac appears to be without clinical significance.

The third group is usually associated with valvular disease of rheumatic origin. It has been suggested that mediastinal adhesions increase the mechanical disability of the myocardium, as at death such hearts are found to weigh 22 per cent more than those of patients dying of valvular disease only (Laws and Levine, 1933). Unfortunately, the surgical treatment of such adhesions by freeing the impediment to the heart's action in systole has not given encouraging results.

The second group, however, is of great importance. The generalized form giving rise to constrictive pericarditis is well known, and is commonly tuberculous in origin: occasionally it is of traumatic (McKusick, 1952) or septic aetiology, but it is stated never to be a sequel of rheumatic disease (White, 1935). The diagnostic and therapeutic problems of constrictive pericarditis are well dealt with in papers by Blalock and Levy (1937), Blalock and Burwell (1941), Holmes Sellors (1946), Andrews, Pickering, and Holmes Sellors (1948), Holman (1951), and Evans and Jackson (1952). Localized thickening and contracture of the pericardium, with its resultant progressive and severe disability, has received but little recognition, and the object of this paper is to discuss chronic localized constrictive pericarditis in the light of four such cases.

CASE REPORTS

Case 1.—R. J., male, aged 16 years, schoolboy, Hospital No. 55/48228. Admitted to hospital under one of us (C. S. D.) on April 20, 1955, with a history of being well until May, 1954, when he entered another institution with a left-sided pleural effusion. When this effusion cleared he was better for a few months, but steadily became more dyspnœic for the six months prior to admission.

The main clinical features on admission were:—

a. Gross hepatomegaly due to obstruction of the inferior vena cava.

b. Slight evidence of obstruction of the superior vena cava—venous pressure in the (right) arm of 8·5 cm. of saline.

c. No evidence of valvular disease of the heart. B.P. 130/80.

d. X-ray film of the chest showed enlarged heart (*Fig*. 702), and screening revealed lack of movement along the right atrial border. This was confirmed by kymography (*Fig*. 703). Cardio-angiography showed a definite filling defect on the right border of the right atrium, otherwise the appearances were normal (*Fig*. 704). Examination of the films taken a year before showed evidence of pericardial effusion.

The diagnosis was considered to be a localized intrapericardial lesion, probably tuberculous, but possibly neoplastic in origin.

OPERATION (May 12) (J. T. C.).—Exposure was obtained by a midline, vertical, sternal split incision. Good ventricular contraction was present and the pericardium was only slightly thickened and adherent over the right ventricle, and it was normal and free over the left ventricle. A tuberculous abscess was found between the pericardium and the right atrium, and a further bilocular abscess inferior and to the left of this, mainly within the wall of the right ventricle. There was also thickening and constriction of the pericardium adjacent to the superior vena cava. The abscesses were unroofed, the wall in places being almost 1 cm. thick, and the adherent pericardium by the superior vena cava was excised. In spite of a perforation of the right ventricle recovery was uneventful, save for a drug dermatitis five weeks later.

Now (January, 1958) he is symptomless, and the liver is of normal size.

The essential features in this case were localized tuberculous abscesses proved by histology, causing a filling defect in the right atrium, and infiltration of the muscle of the right ventricle with marked obstruction of the inferior vena cava.

FIG. 702.—*Case* 1. Note the enlarged heart.

FIG. 703.—*Case* 1. Note the relative lack of pulsation of the lower right cardiac border.

Case 2.—L. T., male, aged 9 years, schoolboy, Hospital No. 55/57291. Admitted to hospital Nov. 18, 1955 under Dr. J. W. Brown, after being in a sanatorium for twenty months. The history was that he developed bilateral tuberculous pleural effusions in 1952. Tuberculous pericarditis followed the subsidence of the pleural effusions in 1953, and in 1954 there occurred the spontaneous discharge through the chest wall of an intrapericardial abscess.

The main clinical features on admission were:—

a. Gross ascites and hepatomegaly (*Fig.* 705).

b. Obstruction of the superior vena cava. On cardiac catheterization the mean right atrial pressures were 22–24 mm. Hg, inspiration 28 mm. Hg, expiration 16 mm. Hg. The catheter could not be passed through the tricuspid valve.

c. No evidence of valvular disease of the heart. B.P. 100/70.

d. Kymography Dec. 7: The heart in the frontal plane shows a slightly abnormal flat-topped wave on the left side of the heart, indicating some delay between the end of diastole and the commencement of systole, and there is also less difference between the speed of the two movements as compared with normal. On the right side the right atrium is represented by a straight line, which indicates an absence of pulsation, and the findings taken together are strongly suggestive of constrictive pericarditis. An oblique kymogram after a barium swallow was also done, but apart from showing a diminished amplitude of pulsation it does not give any further information.

The diagnosis was considered to be constrictive pericarditis, with the main involvement in the right atrial region.

OPERATION (Jan. 2, 1956) (J. T. C.).—Exposure was obtained by a midline, vertical, sternal split incision. The pericardium was exposed easily as both pleural cavities seemed normal as far as could be seen. The pericardium was normal except for a localized area running from the

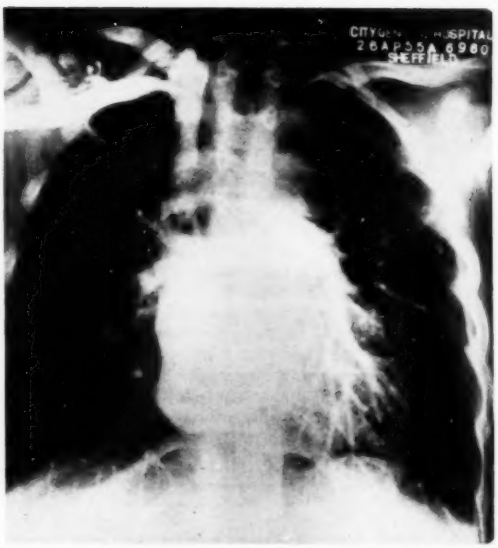

FIG. 704.—*Case* 1. Note the filling defect in the right atrial region.

region of the superior vena cava to the inferior vena cava and over the right atrium.

This firm area was opened and 60 ml. of thick tuberculous pus under pressure was aspirated. The outer and anterior walls of the cavity were excised and the superior and inferior vena cava freed.

The essential feature at this operation was the drainage of a localized intrapericardial tuberculous abscess which was causing obstruction of the superior and inferior vena cava.

Slight improvement only took place and by June the condition was as bad as before operation.

Kymography report June 12: "Further examination after operation still shows a relative absence of movement along the right border of the heart, but there is in fact slightly more movement than prior to the operation. The wave form on the left border had altered slightly and was a little more flat-topped, but otherwise the appearances were unchanged."

SECOND OPERATION (Aug. 3) (J. T. C.).—Exposure was obtained as before.

a. Two firm bands anterior to and kinking the superior vena cava were excised.

FIG. 705.—*Case* 2. Note gross ascitis and scar outside right nipple through which an intrapericardial abscess discharged.

b. A firm mass of adhesions anterior to the inferior vena cava which obstructed it by kinking was sectioned and scar tissue excised.

c. A further mass of caseous matter from the region of the previous abscess was removed.

d. A firm band holding the apex of the heart to the diaphragmatic aspect of the pericardium was sectioned.

The essential features at this operation were the division of fibrous bands constricting the superior vena cava and the inferior vena cava and also holding the apex firmly.

PROGRESS.—August, 1957: He has improved, can run about and play games, but he still has ascites and hepatomegaly, and is far from normal. However, cardiac catheterization (Aug. 15) showed no evidence of abnormal pressure gradients in the inferior vena cava, right atrium, right ventricle, and the pulmonary artery.

Liver puncture biopsy, Aug. 16, showed no abnormality and guinea-pig inoculation of ascitic fluid was negative for tuberculosis (Aug. 16).

Case 3.—A. B., female, aged 6 years (date of birth, Jan. 1, 1946), schoolgirl, Hospital No. 52/10007. Admitted to hospital on Sept. 20, 1952, under Dr. R. R. Gordon, with a week's history of headache, fever, and rapidly increasing dyspnœa. The essential finding was a large pericardial effusion. Two days later this was aspirated by the substernal approach and 90 ml. of straw-coloured fluid, slightly blood-tinged, was removed. Culture and guinea-pig inoculation proved its tuberculous nature. On Sept. 26 aspiration was repeated and 50 ml. of straw-coloured fluid was removed (*Fig*. 706). A month after admission hepatomegaly and ascites were noted, with slight œdema of the legs. She was given a course of streptomycin and P.A.S. for a month, and was eventually discharged on June 19, 1953, still with the signs of obstruction of the inferior vena cava. She was followed both as an in-patient and out-patient until May, 1957. She attended school and was able to get about fairly well in spite of partial obstruction of the inferior vena cava.

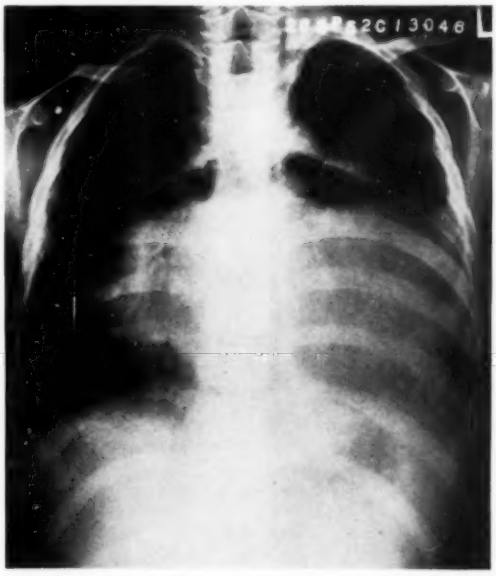

FIG. 706.—*Case* 3. Note the large tuberculous pericardial effusion.

May 17, 1957: I was asked to see her with a view to surgery, which until then had been delayed as she was not typical of generalized constrictive pericarditis.

The essential findings then were:—

a. Œdema lower limbs, gross hepatomegaly, ascites, but no evidence of obstruction of the superior vena cava.

b. X-ray screeening and kymography of the enlarged heart showed moderate pulsation of the left and anterior borders of the heart, but practically no movement in the lower right atrial region (*Figs*. 707, 708).

The diagnosis was considered to be a chronic localized pericardial lesion producing obstruction of the inferior vena cava.

OPERATION (May 28) (J. T. C.).—Exposure was obtained by a midline, vertical, sternal split incision. The pericardium was normal over the left atrium and ventricles, and when opened was free over the ventricles except for two bands near the apex of the heart, which were divided.

The right atrium was freed with difficulty and the scar tissue excised in freeing the inferior vena cava was up to 2 cm. thick.

The essential feature in this operation was the freeing of the inferior vena cava which was buried in dense scar tissue, and the latter on histological examination showed no evidence of tuberculosis.

At the present time (January, 1958) her leg œdema and ascites have disappeared and her liver is much smaller. Her exercise capacity has increased considerably. Cardiac catheterization (Aug. 29, 1957) showed no abnormal pressure gradients in the superior vena cava, inferior vena cava, right atrium, right ventricle, and pulmonary artery.

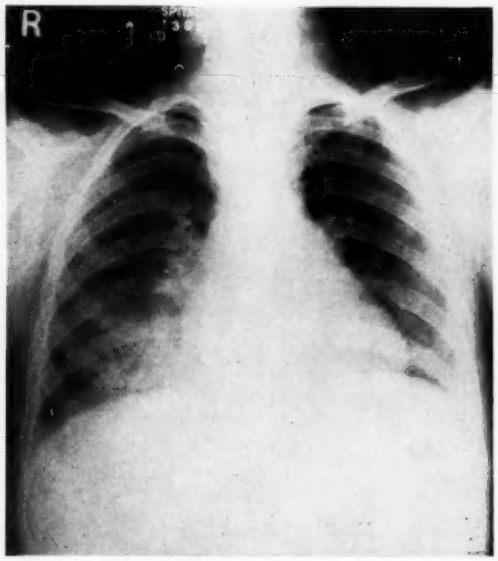

FIG. 707.—*Case* 3. Note the size of the breast five years after the initial tuberculous pericarditis.

FIG. 708.—*Case* 3. Note the lack of pulsation in the lower right atrial region.

Her progress was retarded for several months by a recurrent right-sided pleural effusion which needed tapping on numerous occasions. Guinea-pig inoculation has never shown this to be tuberculous in origin. However, liver puncture biopsy does show that fibrosis is present.

Case 4.—J. B., male, aged 25 years, fruit merchant, Hospital No. 57/74845. The patient was admitted to the Royal Hospital, Sheffield, under Professor C. H. Stuart-Harris from May 10 to June 12, 1956.

HISTORY.—This patient received multiple shrapnel wounds whilst fighting in Korea in 1951. Numerous pieces of shrapnel are still embedded in his chest. Since 1951 he has had spasmodic gripping pains in the chest, but no prolonged illness. During the last four years he has also had attacks of shivering about twice weekly, each one lasting a few hours. He has been investigated for malaria and no parasites were found in the blood. The week before his admission to hospital on May 10 he developed a gripping pain in his chest in front and in between the shoulder-blades. The pain was constant and not related to exertion or meals. He was dyspnœic but did not develop any cough, sputum, or swelling of the ankles. The patient had had no pains in the joints and had no past history of rheumatic fever.

ON ADMISSION.—The main clinical features were:—

a. A friction rub over the whole of the precordium, with the later development of a pericardial effusion. P. 104, B.P. 100/70. E.S.R. 50 mm./hr.

b. Bilateral pleural effusions.

c. Enlargement of the liver 5 cm. below the costal margin.

The response to salicylates was rapid and the patient was discharged with the presumptive diagnosis of acute benign non-specific pericarditis.

A year later the patient was taken ill with fever, lassitude, cough, and sputum, and was admitted on June 5, 1957, ten days later, under Dr. E. G. Rhind. The essential features now were the gross enlargement of the liver and bronchitis. The latter rapidly cleared, but X-ray screening and kymography (*Fig*. 709) showed normal cardiac pulsation except in the region of the right atrium. Investigation for other disease was negative.

The diagnosis was therefore considered to be a chronic localized pericardial lesion producing obstruction of the inferior vena cava, with which Professor C. H. Stuart-Harris agreed.

AT OPERATION (July 8, 1957) (J. T. C.).—Exposure was obtained by a midline, vertical, sternal split incision. No evidence of war injury was found. The pericardium was normal over the left ventricle, slightly thickened over the right ventricle, and only loosely adherent in the region of the superior vena cava. The lower and right borders of the heart showed a succession of small cavities, with fibrosis and scar tissue over 1 cm. thick, in the region of the inferior vena cava. The apex of the heart was also adherent to the diaphragmatic pericardium. The heart was completely freed, together with the inferior vena cava, and the blood pressure rose at once from 110/70 to 130/100 in spite of perforating the right atrium.

The essential feature of this operation was the freeing of the inferior vena cava from dense fibrous tissue, but the point in contradistinction to the previous cases, which were all of proved tuberculous origin, is the histological report on the excised scar tissue:—

"Several fragments of thickened pericardium. Sections show fibrovascular tissue with areas of round-celled

infiltration and perivascular cuffing. Not histologically suggestive of tuberculosis. No acid-fast bacilli seen. The picture suggests a rheumatic origin." (*Fig.* 710).

The hepatomegaly is subsiding and the general condition of the patient is improving (August, 1957). Cirrhosis of liver, however, is present, Jan. 11, 1958.

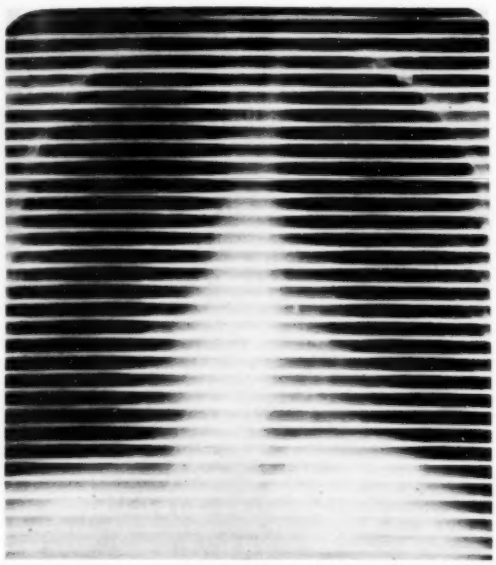

FIG. 709.—*Case* 4. Note the lack of pulsation in the right atrial region.

FIG. 710.—*Case* 4. High- and low-power views of histology of excised scar tissue suggestive of rheumatic origin.

Cardiac catheterization, Oct. 15, 1957, shows no abnormal pressure gradients between the inferior vena cava, right atrium, right ventricle, and pulmonary artery.

DISCUSSION

Aetiology.—The aetiology of the lesion in the first three of the four cases recorded here is undoubtedly a tuberculous infection, and in this it accords with the usual findings in constrictive pericarditis (Andrews and others, 1948). The fourth case, however, apparently followed an attack of acute benign non-specific pericarditis occurring a year previously, though the histological report and plates (*Fig.* 709), which have already been given, suggest either a rheumatic origin or a chronic non-specific inflammatory infiltration.

Carmichael, Sprague, Wyman, and Bland (1951), in a long-term follow-up of 50 cases of acute benign non-specific pericarditis from Boston conclude that constriction does not occur, and they consider that the original diagnosis was wrong in the one case in their series, in which fixity and calcification did take place some four years after the initial attack of pericarditis. Krook (1954), however, in a study of 24 cases of acute benign non-specific pericarditis from Malmö, has followed 2 patients through to the development of constrictive pericarditis. The histology of the excised scar tissue did not suggest any definite cause in either of his 2 cases. Acute benign non-specific pericarditis has been considered a virus infection, a bacterial infection, and a hypersensitive response to unknown allergens akin to rheumatic fever. The fact that in Krook's 2 cases no specific histological evidence of cause was found does not rule out tuberculosis, as in our third case microscopy was of no help in assessing the aetiology, though tubercle bacilli were twice obtained in the pericardial fluid five years earlier. Tuberculosis can be ruled out in our fourth case, as in an area of residual infection in one of the 'abscesses' there is round-celled infiltration and perivascular cuffing, and the scar tissue shows areas similar to rheumatic nodules (*Fig.* 710). It therefore appears that the fourth case in our series developed chronic localized constrictive pericarditis after an infection resembling acute benign non-specific pericarditis, which has given a histological picture not unlike that of rheumatism. Acute rheumatic infection is almost certainly a hypersensitive response, and if the allergens originating this response may themselves be produced by a variety of factors it is possible to conceive that

acute benign non-specific pericarditis and acute rheumatic pericarditis may be nothing more than variations of similar types of infection in different hosts. Further, if one can give rise to constriction (Krook, 1954) why should not the other behave similarly on rare occasions in spite of all that has been written to the contrary? Rheumatic fever does produce scar tissue in the heart valves with stenosis and gross distortion, also it produces scar tissue on the external aspect of the pericardium leading to mediastinopericarditis, so that there is no obvious reason why similarly produced scar tissue in the pericardium should not lead to constriction and distortion.

In conclusion, tuberculosis appears to be the common cause of chronic localized pericarditis, though virus infection, septic infection, trauma, and rheumatic disease can be the aetiological factor. It may well be that both the clinical features and the pathological sequelæ of pericarditis in general need a careful re-appraisal.

Pathology.—All 4 cases, involving five operations, showed localized fibrosis and dense scar tissue, and at three operations there were abscesses present. In 2 there was thick tuberculous pus, and in the fourth case, slightly turbid fluid, which unfortunately was not examined. In the 2 tuberculous cases the abscesses extended deeply into the myocardium, and in the second case this probably caused the difficulty in the passage of the cardiac catheter from the right atrium to the ventricle.

At each operation the lesions were localized, giving rise to an inflow obstruction of the blood to or in the heart by compression of a vena cava or cardiac chamber, and also in three of the five operations there were localized adhesions at the apex, impeding systolic contraction.

Thus, the essential pathological features were a localized obstruction to diastolic filling of the heart to which was added, in some cases, a localized fixity of the apex, preventing adequate systolic contraction of the heart.

Diagnosis.—The diagnosis is suggested by an obstruction to one vena cava, or to one vena cava more than the other, following the history of an attack of pericarditis. In all these cases the inferior vena cava was mainly affected and there was no evidence of valvular or mediastinal disease to account for the condition. Cardiac catheterization and cardio-angiography clinch the diagnosis.

Cardio-angiography showed the localized lesion beautifully in one of our cases (*Case* 1, *Fig.* 704) and cardiac catheterization was suggestive (in retrospect) of it in *Case* 2. Chiche, Carlotti, and Agar (1957) have pointed out the value of cardiac catheterization in localizing the lesions in constrictive pericarditis.

Treatment.—A mechanical obstruction due to fibrosis can only be relieved by surgery, and the optimum time for surgery is directly the condition is proved to be a scar-tissue obstruction. This is suggested by a lack of adequate response to medical treatment. Surgery should take place before even minor degrees of chronic invalidism have sapped morale, wasted time, and given rise to irreparable liver damage. A liver biopsy is worth taking if hepatomegaly and ascites have lasted over two years, as improvement may not take place after that period owing to fibrosis of the liver (Evans and Jackson, 1952).

Kymography and other investigations, as in generalized constrictive pericarditis, will show the area of maximum pathology and indicate the direct approach to the lesion. In these cases a vertical, sternal split incision has given the most direct access, and this approach has the advantage of avoidance of the pleural cavities when intrapericardial tuberculous abscesses may be encountered.

All 4 cases were given adequate therapy for tuberculosis, the first three as they were of proved tuberculous origin, and the fourth in case it was an atypical tuberculous lesion.

Prognosis.—It is too early to give a long-term review of the results of surgery for chronic localized constrictive pericarditis, but there is no reason to suppose that they will be less satisfactory than those of the more generalized forms of constrictive pericarditis. In other words, a good result may be anticipated with adequate surgery in patients without gross myocardial or liver damage, and equally important to note is the converse—that inadequate surgery and delay in sending the patient for surgery gravely prejudices the chances of recovery.

CONCLUSION

The syndrome of chronic localized constrictive pericarditis is discussed in the light of 4 such cases. The essential features are a localized obstruction to the diastolic filling of the heart, to which was added, in 3 cases, a localized fixity of the apex, so impeding adequate myocardial contraction.

Addendum.—A fifth case has been operated upon successfully since this paper was written. R. M., male aged 30, miner, Hospital No. 58/92570, 6½ years history, tuberculous pathology giving localized destruction of the I.V.C., abscess in right ventricular wall, and apical adhesions.

Acknowledgements.—We are indebted to Professor C. H. Stuart-Harris and Dr. J. W. Brown for criticism in preparing this paper; also to the latter for allowing many of the investigations to be carried out in the Sheffield Regional Cardiovascular Centre at the City General Hospital; and to his registrar, Dr. E. C. Gray, for the cardiac catheterization studies. We are grateful to Dr. T. Lodge for the kymography and Dr. A. J. N. Warrack for the pathology of the cases; and Dr. J. Johnston for his skill in anæsthesia.

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AN UNUSUAL CASE OF INTUSSUSCEPTION

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INTUSSUSCEPTION in the adult, though relatively uncommon compared with the condition in childhood, is a well-known cause of 'acute abdomen'. Eliot and Corscaden (1911), in whose classic paper 300 cases are cited; Christopher (1936); Donhausen and Kelly (1950), who collected 665 cases from British and American literature; and Brown and Michels (1952) have all given detailed accounts of the disease in the adult.

A recent case presenting certain unusual clinical and pathological features seems, however, to be worthy of comment.

CASE REPORT

R. R., a 61-year-old dock labourer, for some months previous to the onset of his acute symptoms had noted loss of appetite and had felt off colour. He had no pain, however, and there was no change in his bowel habit.

On Dec. 26, 1957, six days before admission to hospital, he carried out a household removal, and whilst lifting a wardrobe felt a sudden severe pain in his right iliac fossa. The pain eased off gradually and for the next few days, though still present, it did not trouble him unduly. He felt off his food, but he continued to do light work at the docks. The day before admission the pain in the right iliac fossa increased and then quite suddenly it travelled round and settled in the left side of the abdomen. There it persisted with variable intensity throughout the night, attacks lasting twenty minutes to three-quarters of an hour, till he was sent in to hospital on Jan. 1, 1958.

During this period there had been no change in the bowel habit, the bowels having opened normally on the day before admission, and there had been no vomiting. The patient also thought that his urine had been blood-stained on a number of occasions during the previous two days. He had no other urinary symptoms.

ON EXAMINATION in hospital, R. was seen to be a pale, ill-looking man. Pulse, 128/min.; temperature, 102° F.; blood-pressure, 130/90. The abdomen, which was not distended, was markedly tender on the left side over an ill-defined palpable mass extending from the left iliac crest up towards the left costal margin and backwards into the left loin. There was no generalized tenderness and bowel-sounds were normal. Rectal examination was negative. Microscopical examination of the urine showed numerous red cells; culture of the urine was later shown to reveal only a scanty growth of coagulase-negative staphylococci. Diagnosis was considered to lie between diverticulitis of the descending colon and infected hydronephrosis, perinephric abscess, or a renal tumour.

The patient settled well after admission, the pain lessened, and the temperature fell to 98·8° F. The following day, however (Jan. 2), the pain increased and for the first time he vomited, though only a small amount. He vomited twice, again small amounts, the next day (Jan. 3) and the pain became severe. He continued to pass flatus per rectum. It was now possible to feel a hard well-defined lower pole to the mass originally noted, but the full extent of the mass was obscured by guarding. An intravenous pyelographic examination was carried out; this showed normal pyelograms, with both kidneys excreting.

The increase of the patient's symptoms, and some general deterioration in his condition, with the increased prominence of the palpable mass in the abdomen, indicated exploration, and this was performed on the evening of Jan. 3.

AT OPERATION.—Anæsthesia administered by Dr. I. MacIntyre was induced by 0·4 g. of half-strength thiopentane intravenously and maintained by cyclopropane and oxygen in closed circuit from a Gillies machine, with 120 mg. of flaxedil as a relaxant. An oblique muscle-cutting incision was made in the left flank,

FIG. 711.—Drawing illustrating the condition seen at operation. The sheath or intussuscipiens has sloughed and the intussusceptum is visible with a nodular carcinoma at its apex.

overlying the palpable mass. The tissues were noted to be œdematous and the exploring finger went easily through the peritoneum into what appeared to be a large abscess cavity. The cavity, however, contained large quantities of green mucinous material and gas instead of pus; and lying exposed in it was a large sausage-like object, about 16 cm. long, covered with mucous membrane (*Fig.* 711). Further exploration showed that this protruded through a large defect in the descending colon, 5 cm. below the splenic flexure. This object lifted up easily, and a narrow band of the posterior wall of the colon was seen to lie posterior to it, extending down to the left iliac fossa, and there joining the lower part of the descending colon,

where the circumference of the bowel became continuous again. It was then recognized that the condition was an intussusception; the sheath or intussuscipiens had evidently sloughed for 16 cm. in the descending colon, and the oblong mass was the intussusceptum. The intussusceptum itself, though œdematous and congested, was viable and appeared to be clothed with colonic mucosa. At the apex was a nodular ulcerated area about 3 cm. in diameter, with the appearance of a carcinoma.

After suction of the escaped mucus, and swabbing the area with dettol swabs, the general peritoneal cavity was easily entered by the separation of light adhesions, and the intussusception was traced round to the hepatic flexure, where the ileum was seen to enter the reefed-up colon. The small intestine was only moderately distended. An attempt to reduce the intussusception was unsuccessful and it was felt that there was no alternative but to proceed to radical measures. From the lower part of the oblique incision a vertical left paramedian incision was extended upwards. The intussusception, comprising the terminal ileum, cæcum, ascending, transverse, and most of the descending colon, was then resected in continuity. The distal part of the ileum and the proximal end of the distal colon, divided through healthy colonic wall 2 cm. below the gaping hole in the lower part of the descending colon, was then stitched together for 8 cm. to form a spur, and brought out in the left iliac fossa through the lower end of the oblique incision, with Paul's tubes inserted into both ends, following the usual Paul Mikulicz technique (Maingot, 1948). At the end of the operation, which lasted 1½ hr., during which 800 ml. of blood were given, the patient was in good condition, the blood-pressure being 140/80.

PROGRESS.—He made a satisfactory recovery. He had 500 mg. terramycin intravenously twice a day for three days, followed by 500 mg. by mouth four times a day for eight days. The ileostomy acted well. An enterotribe was applied to the spur on Jan. 17. The skin had become somewhat excoriated around the bowel opening, and on Feb. 18 the ileocolostomy was closed extraperitoneally. A small quantity of fæces continued to be discharged through a tiny opening in the wound, though the bowels moved normally. On May 15, this opening was explored. It was found that dense scar tissue had prevented a complete closure of the opening in the mucous membrane. The track between the skin and the intestine with the surrounding scar was excised, the opening in the bowel wall was closed, and the abdominal wall repaired. The wound healed soundly, and the patient went home on June 2.

THE SPECIMEN.—The microscopical report on a section of the specimen was given by Professor E. C. Mekie, Conservator of the Royal College of Surgeons Museum, as follows (Feb. 3): Section from the apex of the intussusceptum shows a papilliferous adenocarcinoma with considerable differentiation and in the section studied, very little infiltration. The picture is grossly distorted by marked œdema, presumably due to the mechanical obstructions of the blood-supply occasioned by the intussusception.

DISCUSSION

Two special features require discussion in this case: (1) the clinical features; and (2) the pathological findings.

The Clinical Features.—The cardinal symptoms of intussusception in both adults and children are: (1) periodic attacks of abdominal pain; (2) vomiting; (3) a palpable tumour in the abdomen; and (4) passage of blood per rectum. (Brown and Michels, 1952; Wangensteen, 1955.)

Eliot and Corscaden (1911), however, state that the clinical picture in intussusception in the adult is by no means constant; and other authorities have noted that the picture is less characteristic in the adult than in the child (Thomson, Miles, and Wilkie, 1931). Brown and Michels write, "The diagnosis of intussusception in the adult is frequently difficult", and according to Donhauser and Kelly (1950) the symptomatology in cases of intussusception in the adult is so indefinable that a definite diagnosis can be made only rarely. Cases which have simulated other lesions such as cholecystitis or peptic ulcer have been described (Christopher, 1936). Moreover, in contrast to other forms of bowel strangulation, intussusception, at least in the earlier phases, may occur without localized tenderness or muscle spasm owing to the fact that the strangulated segment, the intussusceptum, is ensheathed by the intussuscipiens, which thereby protects the parietal peritoneum from irritation (Nichols, 1941).

Eliot and Corscaden state that those intussusceptions due to malignant tumours may present in one of three ways: (1) An acute onset in a previously healthy patient (uncommon); (2) A history of a primary growth in a distant part of the body (usually a sarcoma) precedes the intussusception; (3) A history of chronic obstructive symptoms, intussusception being suggested by the presence of a palpable mass. This is the commonest type.

There may be varying degrees of intussusception—acute, subacute, and chronic—there may therefore be a history suggestive of recurrent attacks of bowel obstruction, symptoms being present for a period of days or years. In Brown and Michels's (1952) 15 cases the average duration of symptoms was eight months.

The striking clinical feature of the present case is the onset of pain in the right iliac fossa, where the intussusception originated, followed by a clear history of the pain travelling round to the left side of the abdomen. This travelling of pain in intussusception has been noted by Wangensteen (1955), and there can be little doubt that the pain in the present case was caused by the actual development of the intussusception. A muscular strain may start the onset of intussusception, as Eliot and Corscaden (1911) have described, and this had occurred in the present case also; but the significance of this feature was not appreciated at the time.

When the mass developed in the left groin, intussusception was still not considered; the transient hæmaturia focused attention on the kidney as a possible cause of the swelling, the pyrexia being considered consistent with a renal condition. Absence of obstructive bowel symptoms till the day of operation was also misleading, there being at no time the characteristic blood or mucus in the stools. Intussusception not being suspected, Dance's sign—emptiness of the right lower quadrant of the abdomen (Ferrer, 1950)—was not sought after. When operation was eventually decided upon, the diagnosis rested tentatively between (1) diverticulitis with paracolic abscess, (2) carcinoma of the colon with paracolic abscess, and (3) perinephric abscess.

Pathology.—The pathology of intussusception is well described by many authors. The great percentage of primary intussusceptions in the adult are of the ileo-ileal, ileocæcal, and ileocolic types (Donhauser and Kelly, 1950), and the present case

falls into the category of ileocolic, or perhaps more correctly colocolic.

In the majority of cases the intussusception is single, and descending in direction, though more complicated types have been described (Walton, 1911). In the adult, intussusception is usually due to an organic cause. In Eliot and Corscaden's (1911) series there were 60 instances of benign tumour and 40 of malignant tumour. In Donhauser and Kelly's series (1950) malignant tumours were the cause of the intussusception in 20 per cent of the whole series.

The obstruction in intussusception is afforded by the active contraction of the sheath, especially at its neck. An actual block of the lumen does not exist. The continuity of the intestinal lumen between proximal and distal segments remains intact, and in chronic intussusceptions in which the bowel wall changes are not as marked, the intestinal current is preserved. The greatest alteration in the bowel in acute intussusception occurs in the intussusceptum (Wangensteen). The vessels in the mesentery becoming constricted, the intussusceptum and the reflected layer become œdematous and swollen, and if the constriction at the neck of the intussuscipiens is tight, the circulation in the intussusceptum may be entirely arrested and gangrene may ensue (Watson-Cheyne and Burghard, 1913).

If untreated, the condition progresses; the gangrenous intussusceptum becomes a slough. The spontaneous evacuation of this gangrenous intussusceptum, composed of varying lengths of small intestine, cæcum, and colon, has been frequently described, particularly in older literature (Hutchinson, 1874, quoted by Close, 1931; Gould and Pyle, 1897; Eliot and Corscaden, 1911; Nichols, 1941). In the days before routine abdominal surgery such cases were thus occasionally cured spontaneously, though some died later of obstruction due to stenosis of the bowel.

The intussuscipiens, or sheath, on the contrary, rarely shows significant gross changes (Walton, 1911; Bockus, 1944; Wangensteen, 1955). It is not infrequently congested, or it may exhibit deposits of fibrin or other evidence of local peritonitis. Wangensteen observes that it may occasionally even perforate consequent upon pressure of the invaginated bowel upon it. In Eliot and Corscaden's 300 cases it is recorded that in one the intussuscipiens was in a condition of incipient gangrene, and in a second (Case 223) at operation the intussuscipiens was found split; but we have been unable to find a reference to gross sloughing of the intussuscipiens similar to that in the present case. We consider that the cause of this necrosis of the ensheathing bowel was probably the pressure of the bulky intussusceptum producing an interference of the blood-supply of the colon wall; the colon, in contrast with the small intestine, being a comparatively avascular structure (Ross, 1952). The intussusceptum, though congested, was quite viable; the sloughing of the enclosing sheath evidently relieved pressure on it, though it was still tightly gripped proximally, and eventually would have become gangrenous.

That this sloughing of the sheath for at least 16 cm. should be unassociated with gross contamination of the peritoneal cavity is a remarkable feature of the present case. The explanation would appear to be, first, that the colon had emptied itself after the process of intussusception began, as shown by the continuation of normal bowel movements up to the day before admission. Secondly, the intussusceptum, continuing to swell after the sheath had burst, became so tightly gripped as it passed through the bowel at the upper end of the tear that no further fæcal matter passed through. The clinical history and operative findings suggest that complete obstruction was of only short duration.

The operative treatment requires no special comment. We considered that there was no alternative to the radical procedure adopted, which fortunately proved successful.

SUMMARY

A case of intussusception of the colon in a man aged 61 is described with: (1) Unusual clinical features; (2) Gross pathological changes. The sheath had sloughed for 16 cm., leaving the intussusceptum free in the abdominal cavity, but with minimal soiling of the peritoneum.

Resection of the affected intestine proved successful.

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SOLITARY ENDOBRONCHIAL METASTASIS FROM CARCINOMA OF KIDNEY

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METASTASES from renal carcinomata notoriously occur in bizarre sites, but a solitary endobronchial deposit, mimicking a primary carcinoma of bronchus and occurring several years after the removal of the primary growth, does not appear to have been reported previously.

CASE REPORT

An engine driver, aged 50 years, was first seen in 1950 with hæmaturia. A right nephrectomy was performed for clear-cell carcinoma of characteristic appearances (*Figs.* 712, 713). He was well up to March, 1954, when he had a hæmoptysis. Bronchoscopy showed a polypoid mass in the left main bronchus, biopsy from which caused very furious bleeding (estimated at 1 pint). Section showed only fragments of connective tissue and hyaline cartilage. Repeat bronchoscopy showed only some thickening of the mucosa of the left stem bronchus between the posterior and middle basal segmental bronchi but no tumour.

Thereafter he remained symptom free until November, 1957, when he had a further hæmoptysis. He felt very well and described himself as "in the pink of condition". This was perhaps truer than he realized, as he had polycythæmia vera! On examination, he was very plethoric, with clubbing of his fingers and a palpable spleen. His chest was rather fixed and emphysematous, but there were no added sounds. Hb 22·6 g. per 100 ml. (152 per cent), R.B.C.s 8·0 million per c.mm., P.C.V. 64 per cent, W.B.C.s 9000 per c.mm., platelets 720,000 per c.mm., E.S.R. 2 mm. in 1 hr. (Wintrobe).

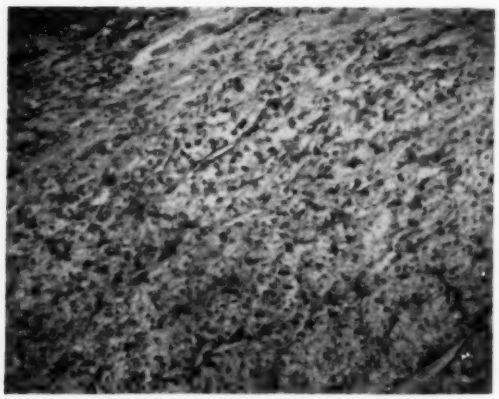

FIG. 712.—Clear-cell carcinoma of kidney. Hæmatoxylin and eosin. (×100.)

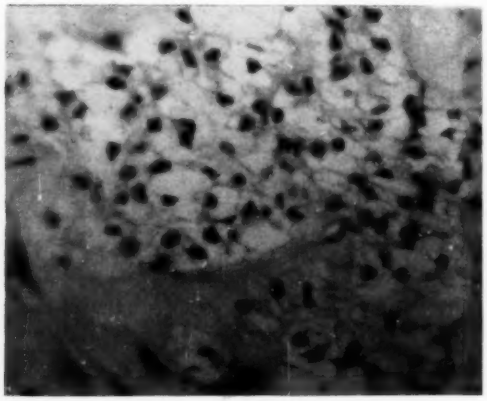

FIG. 713.—Clear-cell carcinoma of kidney. Hæmatoxylin and eosin. (×440.)

FIG. 714.—Metastatic clear-cell carcinoma in bronchus. Hæmatoxylin and eosin. (×100.)

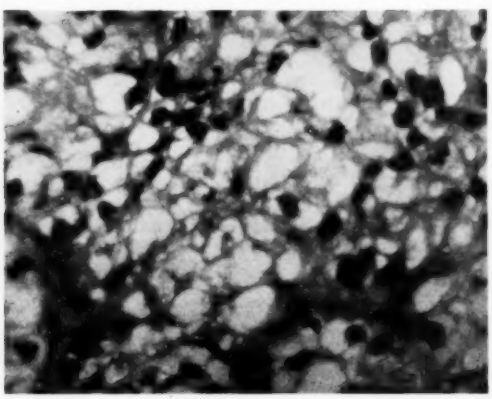

FIG. 715.—Metastatic clear-cell carcinoma in bronchus. Hæmatoxylin and eosin. (×440.)

Bronchoscopy (Dec. 3, 1957, Mr. T. W. Stephens) showed a firm whitish mass resembling slough loosely attached to the medial wall of the lower stem bronchus. This was removed to expose an area on the bronchus which looked like granulation tissue. A biopsy was taken,

but showed only necrotic tissue. He was bronchoscoped again on March 11, 1958, by Mr. Stephens and the same appearance of a small granulomatous mass with slough was seen. The slough was removed with biopsy forceps and a specimen obtained from the depth of the apparent tumour.

Biopsy Specimen.—Section shows a clear-cell carcinoma frankly invading bronchus (*Figs.* 714, 715). The cells are large, with abundant, pale, foamy cytoplasm and arranged in solid sheets with thin supporting stroma including thin-walled blood-vessels. The slides were shown to several experienced morbid anatomists (Drs. A. B. Bratton, W. Pagel, S. Robinson, and P. M. Peters), all of whom, quite independently and without knowledge of the history, said that the tumour is indistinguishable from a typical clear-cell carcinoma of the kidney.

The patient remains feeling well and free from symptoms. Radiographs of chest, tomograms, and a left bronchogram are essentially normal and there is no evidence of a second primary carcinoma in the remaining kidney.

DISCUSSION

There can be no doubt that the biopsy specimen is a metastasis from a renal carcinoma. One occasionally sees in primary bronchial tumours small foci of large clear cells. These, however, contain glycogen (Pagel, 1958, personal communication) and invariably merge with areas of unequivocal squamous growth. There are no such areas in this case and the periodic acid-Schiff stain is negative for glycogen. Sometimes metastatic deposits in hilar lymph-glands may ulcerate through the bronchus, but these are associated with deposits in the pulmonary parenchyma (Willis, 1953), of which there is absolutely no evidence in this case despite very thorough clinical and radiological investigation.

Renal carcinomata are very fickle and unpredictable in their behaviour, and metastatic growths have been known to appear in unusual situations such as skin, muscle, tonsils, oral and nasal mucosa, larynx, and iris (Willis, 1953). Endobronchial metastasis does not appear to have been reported previously.

Wintrobe (1956) says that hæmoptysis may occur as a symptom of polycythæmia. But it is an unusual symptom and the possibility of a coexisting bronchial tumour must be considered in all such patients. One should not be led astray by the 'red herring' of the blood dyscrasia !

SUMMARY

A case is described of a 57-year-old polycythæmic man with recurrent hæmoptysis due to an endobronchial tumour histologically indistinguishable from a clear-cell carcinoma of kidney and occurring nearly eight years after nephrectomy for hypernephroma. The diagnosis is discussed and the importance of hæmoptysis as a symptom of bronchial tumour, even in the presence of a blood dyscrasia, is stressed.

I am grateful to my colleagues named for their opinions; Dr. W. E. Clarke for the case-papers and encouragement to publish; Dr. J. T. Prendiville for slides of the kidney; Mr. H. S. Cox for the microphotographs; and Mrs. Cynthia Cook for the typescript.

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ALKALOSIS OF ALIMENTARY ORIGIN IN SURGICAL PATIENTS

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NORMAL cellular function depends on the preservation of a suitable environment for the cells by the maintenance of a large enough volume of extracellular fluid at a constant *p*H. The composition of the extracellular fluid is subjected to repeated disturbances, by the addition of the products of digestion and of cellular metabolism, and by the removal of the components of the digestive secretions, of urine, and of sweat. Several independent mechanisms are present in the body to maintain the acid-base balance, since for the continuance of life the *p*H must be kept within the limits 7·0 and 7·8. In health the variation is slight (7·35–7·45). Any tendency to deviation in *p*H is quickly and effectively combated by the activity of the buffer systems of the blood, altered respiratory function, and the differential excretory powers of the kidney. The buffer systems of the body include hæmoglobin, protein, and the salts of certain weak acids. One of the most important body buffers is the bicarbonate–carbonic-acid system. In every 100 ml. of blood there are 3 ml. of carbon dioxide in the form of carbonic acid and 60 ml. of bound carbon dioxide in the form of bicarbonate. The *p*H depends on the relation

$$\frac{\text{Free } CO_2}{\text{Bound } CO_2} \text{ or } \frac{\text{Acid}}{\text{Base}}$$

which should normally be 1/20. By varying the rate and depth of respiration the body is able to eliminate carbon dioxide more or less, and thereby keep this ratio within its normal limits. When this and other mechanisms such as the renal excretion of acid or basic salts fail, an uncompensated alkalosis results.

Most commonly it follows vomiting in pyloric obstruction. It may be accentuated by gastric lavage. It is often severe in patients who have taken large amounts of alkali.

The four cases reported here illustrate these aetiological factors. All were cases of severe pyloric stenosis. In the first two, copious vomiting was the

main factor; in the third, gastric lavage contributed; in the fourth, excessive consumption of alkalis prevailed.

CASE REPORTS

Case 1.—M. H., male, 62 years, was unconscious on admission to hospital. A history was obtained by proxy. He was known to have had typical ulcer dyspepsia for many years and a recent barium-meal examination had shown evidence of pyloric stenosis. He had suffered from

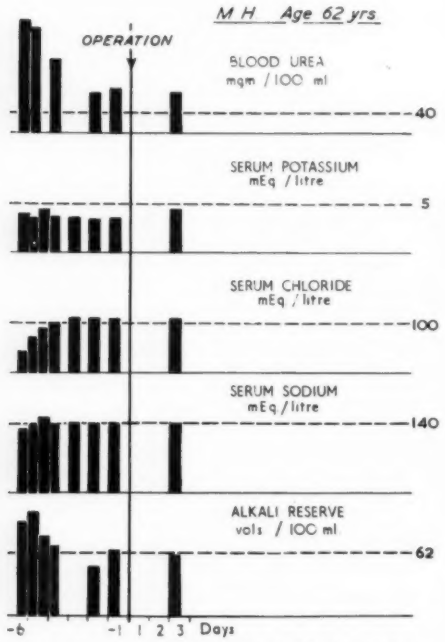

FIG. 716.—*Case* 1. Record of values for blood-urea, plasma-potassium, plasma-chloride, and plasma-sodium, with alkali reserve. The values are mostly taken from the pre-operative, alkalotic period.

intermittent vomiting, much more frequent and copious in the three weeks before admission, culminating in 'black-outs' of 5–10 minutes' duration and mental disorientation on the day prior to admission. He could not be roused from his sleep on the day of admission. He had not passed urine for at least 12 hours.

On examination he was comatose, but with periods of restlessness in which he responded to sensory stimuli. There was marked evidence of dehydration. Respiration was shallow and phasic. Photophobia was present with slight nuchal rigidity. There was no unequivocal evidence of tetany. On abdominal examination the outline of a distended stomach was clearly visible and there was marked succussion but no visible gastric peristalsis.

The provisional diagnosis was pyloric stenosis with alkalosis and treatment was begun by giving intravenous fluid: 2 l. of normal saline were given in the first 4 hr.; 10 l. in the first 36 hr., together with approximately 5 g. of potassium. Gastro-enterostomy was performed after full recovery on the sixth day after admission. The diagnosis was in the meantime confirmed by biochemical examination (*Fig.* 716). The alkali reserve was over 100 vol. per cent at the first reading and the blood-urea was 225 mg. per cent. The plasma electrolytes were all on the low side of normal, the sodium and chloride being more depressed than the potassium levels.

The CO_2-combining power remained high (80 vol. per cent) until the third day, and the blood-urea during this period was 120 mg. per cent. Correction of these severe biochemical changes took three days.

As regards the patient's further progress, three hours after treatment had begun the level of consciousness improved and he was able to answer simple questions. Thereafter there was a relapse into semi-coma, followed by a further improvement twelve hours later, by which time he was conscious but disorientated. On the third day he was mentally alert and there was no further relapse. Urine was passed six hours after admission. The first specimen was alkaline and contained protein and hyaline casts.

Nine months after operation this patient was quite symptom-free. His urine, however, still contained albumin and casts, and his renal urea-clearance was just below 70 per cent of normal.

Case 2.—A. L., male, 55 years. This patient had complained of ulcer symptoms for eighteen years and a duodenal ulcer had been demonstrated radiologically five years prior to admission. Following a long remission he had had a recurrence of symptoms three weeks before admission to hospital, with vomiting two or three times each day. He had noticed that the vomitus contained food consumed at least twelve hours previously. The consumption of alkaline powders had not been unusually excessive. He was ill-looking and dehydrated, with evidence of loss of weight. He had marked air hunger and Trousseau's test was strongly positive. The outline of the stomach was visible and there was a succussion splash, but no visible peristalsis. At the time of examination he was having carpopedal spasms and becoming drowsy. Biochemical examination confirmed the diagnosis; alkali reserve 88 vol. per cent; blood-urea 76 mg. per cent; plasma-sodium 141 m.eq./l.; plasma chloride 65 m.eq./l.; plasma potassium 3·5 m.eq./l.; serum-calcium 9·4 m.eq./l. The urine was acid in reaction, there was proteinuria, sp.gr. was 1022, and there were hyaline casts.

In view of the incipient tetany, 30 ml. of 10 per cent calcium gluconate were administered intravenously, followed by 3 l. of normal saline. He improved on this therapy and became alert, though still complaining of upper abdominal pains in spite of continuous gastric aspiration. Twenty-four hours later the duodenal ulcer perforated. At operation pyloric stenosis and an enormously dilated stomach were found and a Polya gastrectomy was performed. The blood-urea one month later was 48 mg. per cent; the water concentration test gave a sp.gr. of 1015; water dilution test gave a sp.gr. of 1002 and the urea-clearance was 68 per cent in the first hour. There was proteinuria, but there were no casts present.

Case 3.—A. T., male, 44 years, complained of epigastric discomfort of three weeks' duration with repeated vomiting. He complained of loss of weight and energy, of heartburn, flatulence, and anorexia. A diagnosis of pyloric obstruction was made and 2½ l. of foul gastric content was removed by aspiration. Subsequently, operation revealed a carcinoma of the pyloric antrum and a Billroth gastrectomy was performed. Gastric suction was continued for the first two post-operative days, glucose-saline replacement being given intravenously.

During the second day it was noticed that he was becoming drowsy and potassium deficiency was suspected. Biochemical studies at this stage (*Fig.* 717) showed a fairly severe alkalosis, CO_2 combining power 88 vol. per cent; blood-urea 164 mg./100 ml.; and a hypochloræmia of marked degree (78 m.eq./l.). There was also a marked deficiency of potassium and sodium in the serum. In spite of treatment with fluid and electrolyte, he lapsed into

coma and later manifested frank tetany. Fluid was replaced, first 1 l. of normal saline, then 2 l. of 3 per cent potassium solution, and later 3 l. more of normal saline; 10 ml. of 5 per cent gluconate was given at the commencement of treatment. Three days later the serum-electrolyte levels had been restored to normal, the blood-urea alone remaining elevated (*Fig.* 717).

This patient, in spite of a severe extracellular alkalosis, had an acid urine (*p*H 5·6), with proteinuria and hyaline casts. On recovery from coma the urine retained a fixed

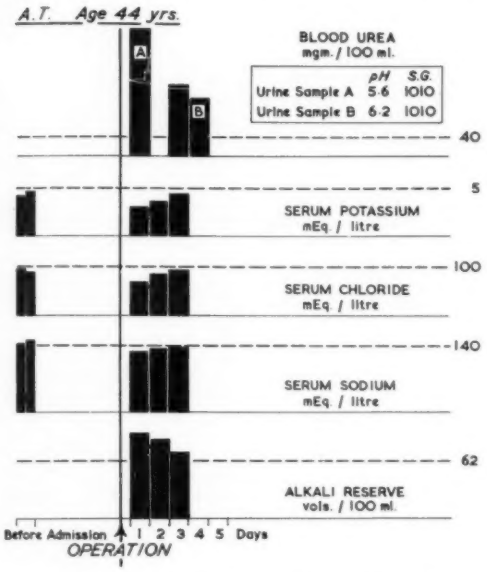

FIG. 717.—*Case* 3. Record of values for blood-urea, plasma-potassium, plasma-chloride, and plasma-sodium, with alkali reserve. Urine samples at A and B had the features shown on the inset. The values are mostly taken from the post-operative, alkalotic period.

sp.gr. of 1010. Unfortunately, after correction of the metabolic disturbance and before improvement in renal function could be assessed, the patient had a secondary hæmorrhage on the seventh post-operative day and died in spite of repeated blood transfusion. Post-mortem examination revealed evidence of a blood dyscrasia, induced by dextran infusions, and acute tubular necrosis; there was marked tubular degeneration, with a hæmorrhagic cellular infiltrate. Some proliferation of the stroma was evident. The glomeruli were normally preserved and there was no periglomerular fibrosis which might have been associated with the complication of pyelonephrosis.

Case 4.—P. L., male, 48 years, had complained of dyspepsia for ten years and had customarily taken sodium bicarbonate for relief. In the three weeks prior to admission he had taken 7 oz. of a proprietary powder containing calcium carbonate, heavy magnesium oxide, sodium bicarbonate, and bismuth carbonate each week, together with two or three teaspoonfuls of sodium bicarbonate before and after each meal. Two days before admission he had had more pain, had vomited repeatedly, and had consumed large quantities of his powders. He became restless, lapsed into delirium, and lost consciousness.

Physical examination revealed marked dehydration, with slow shallow breathing and cyanosis. He had two attacks in which he became rigid, with stertorous breathing. His upper abdomen was distended, but peristalsis was not seen. Trousseau's sign was equivocal and Chvostek's was negative. The urine contained protein together with numerous hyaline and blood casts; the reaction was acid.

Treatment with intravenous saline was begun shortly after admission. He was given 4 l. of saline intravenously in the first 24 hr., thereafter continuing on half-strength saline by mouth. The blood-chemistry findings were: alkali reserve 112 vol. per cent; plasma-chlorides 42 m.eq./l.; blood-urea 150 mg./100 ml. The plasma-chloride rose to 110 by the end of the second day and the urinary chloride, absent initially, rose to 104 m.eq./l. The blood-urea slowly fell to 50 mg./100 ml. after twelve days. Gastro-enterostomy was performed for pyloric stenosis a fortnight after admission. After a month his blood-urea was 50 mg./100 ml., urinary specific gravity was fixed at 1010, and his urea-clearance was only 50 per cent. He was symptom-free, however, had put on weight, and was due to recommence work. After one year his blood-urea was 42 mg./100 ml. and his urea-clearance was 68 per cent. The sp.gr. of his urine was 1012, and there was still proteinuria but no casts.

DISCUSSION

Alkalosis in surgical patients is commonly metabolic, non-gaseous in origin, the result of excessive loss of acid, often combined with an increased intake of alkali. It is commonly due to excessive vomiting in pyloric obstruction, aggravated sometimes by gastric aspiration, in patients taking large doses of alkali (Wilkinson, 1955). Four cases have been described. All showed a high alkali reserve, with lowered plasma-chloride and with sodium and potassium levels which were only a little reduced. It is likely that if the values for the individual blood samples are only slightly lowered, the total loss of electrolytes from the body must nevertheless have been considerable, since hæmoconcentration tends to minimize the reduction.

Three of these patients presented the paradox that, despite the alkalosis, the urine was acid (the fourth was incontinent on admission and no urine could be obtained prior to treatment). This is explained by the fact that, as potassium is lost from the cell, it is replaced by hydrogen ions so that the extracellular alkalosis is accompanied by an intracellular acidosis (Cooke, Segar, Cheek, Colville, and Darrow, 1952; Black and Milne, 1952). In the renal tubule sodium is normally conserved, and is reabsorbed in exchange for potassium and hydrogen; when potassium, however, is depleted its place is taken by hydrogen ions, so that the urine becomes acid. This concept of ion exchange explains the rapid clinical improvement which takes place in those cases of alkalosis with marked potassium depletion when potassium replacing solutions are administered; potassium is returned to the cell and the hydrogen ions revert to the extracellular space, with consequent reduction of the alkalosis.

Metabolic alkalosis can be compensated by increasing the blood carbonic acid as a result of diminished respiratory activity. Bicarbonate is excreted in excess in the tubule, and several other general factors may be at work, such as the cessation of secretion of gastric hydrochloric acid, perhaps induced by potassium deficiency (Carone and Cooke, 1953). Starvation leading to ketonæmia, or poor renal function with accumulation of acid end-products of metabolism, may induce slight correction of *p*H.

Important limiting factors, however, diminish the efficiency of the compensatory mechanism; a rise in blood carbon dioxide for example, tends to increase the bicarbonate reabsorption in the renal tubule (Brazeau and Gilman, 1953). Furthermore there is no easy way of correcting the potassium loss by natural means. The tubular cells are unable to retain potassium ions instead of hydrogen ions (Berliner, Kennedy, and Orloff, 1951) and it is possible that any renal damage incurred increases this loss (Brown, Currens, and Marchand, 1944).

The association of renal damage with alkalosis is a fascinating but complex problem. In all the 4 cases reported there was evidence of marked renal damage. In addition to the high blood-urea (which might in part be due to oliguria and dehydration) the urine was of fixed specific gravity and contained casts. In the 3 surviving patients there was continued evidence of renal damage during convalescence. Two views have been held: on the one hand it has been claimed that the renal damage antedates and predisposes to the alkalosis (Hurst, Houghton, Venables, and Lloyd, 1926); on the other hand, that the alkalosis may be the cause of renal damage. Cooke (1932) stated that there was no convincing evidence of previous renal damage in most reported cases, although there was every reason to suspect it as a possible aggravating factor. It seems clear from the work of Sanderson (1954) that the administration of alkali alone produces alkalosis with difficulty and does not of itself cause renal damage; he found that only a very mild degree of alkalosis developed in a man given 140 g. sodium bicarbonate in 3 l. of milk per day for 21 days. It is known that potassium deficiency produced experimentally in animals can cause renal damage (Follis, Orent-Keilas, and McCollum, 1942), and a similar process is thought to underlie the renal lesion which develops in the human subject in primary aldosteronism (Conn, 1955). Two features in the cases reported here support the view that alkalosis, combined with dehydration and potassium loss, is the cause of the renal damage rather than pre-existing renal disease. In the fatal case (*Case* 3) post-mortem examination showed severe tubular damage of recent origin with no evidence of pre-existing glomerulonephritis; in the surviving cases there has been some evidence that the trend is one of progressive improvement in renal function.

In the treatment of this disorder intravenous administration of sodium chloride solution holds pride of place. This relieves the extracellular dehydration and replaces chloride ions, but has the disadvantage of ignoring the grave potassium loss. The use of ammonium chloride as a source of chloride ions must be avoided in the presence of renal damage, since its efficacy is dependent on the conversion of ammonia to urea in the renal tubule; in addition it might worsen the intracellular acidosis (Cooke and Crowley, 1953). When the urinary output has been re-established the deficit of potassium may be made good by intravenous potassium solution, taking due care to avoid hyperkalæmia at all stages. Finally, since the biochemical balance on intravenous therapy is always precarious, it is important to resume normal feeding as soon as possible. As a necessary preliminary the pyloric lesion must be removed, or, if a simple stenosis, at least by-passed by gastrojejunostomy, and this should be done as soon as the patient's condition permits.

SUMMARY

1. Four cases of alimentary alkalosis are described with severe biochemical changes, tetany, or coma and paradoxical aciduria.

2. The aetiological factors were excessive vomiting from pyloric obstruction, gastric aspiration, or chronic overdosage with alkaline powders.

3. Renal damage was found in each case, and evidence of this remained in the recovery period.

4. The mode of production of the electrolyte disturbance and its effect on the acid-base balance and on the urine acidity are briefly discussed.

We should like to thank Professor C. F. W. Illingworth and Mr. A. W. Kay, in whose wards these patients were treated, for their encouragement and advice in writing this paper. We acknowledge with thanks the help received from the staff of the Biochemistry Department of the Western Infirmary, Glasgow.

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SMOOTH-MUSCLE TUMOURS OF THE ALIMENTARY TRACT

By A. W. BRUCE and A. L. STALKER, Aberdeen

SMOOTH-MUSCLE tumours of the alimentary tract form an unusual and interesting group of neoplasms. Reviews, with case reports, appear mainly in the American literature; these tend to be regional in their scope, or to include lesions found at post-mortem examination or incidentally at operation, and so, we believe, do not give a true picture of the clinical frequency of these tumours. This belief has prompted a study of all leiomyomatous tumours of the alimentary tract in the Aberdeen area in the period 1937–57. Cases found incidentally at operation or at autopsy have been excluded from consideration. The analysis has thus been made on cases presenting with symptoms directly referable to a leiomyomatous tumour (save in one instance in which a large leiomyosarcoma of the stomach coincided with a colonic carcinoma and could barely be considered as an incidental lesion on account of its size and proved histological appearance).

Some 25 cases are available for study—1 in the œsophagus, 7 in the stomach, 11 in the small intestine, and 6 in the rectum. There was no example in the colon. In estimating the frequency of these tumours we have been hampered by lack of precise data on the incidence of the common carcinomata of these sites, but, basing our calculation on the data available, we estimate that during the period reviewed there occurred some 1000 gastric cancers, 960 colonic cancers, and 620 rectal cancers, while about 10–15 cancers of the small intestine were observed. The relative incidence of leiomyomatous tumours is thus extremely low, except in the small intestine where it is apparently about as common as the carcinoma.

In considering the literature on this subject we wish to refer only to those papers which present the most comprehensive and authoritative information, omitting many smaller reviews and case reports. Golden and Stout (1941) give the best general account in the literature, and remark on the high incidence of gastric lesions. Basing their analysis on 1018 cases, derived from 400 publications, and 60 cases of their own, they give the incidence in the various sites as follows: stomach 61·5 per cent, small intestine 19 per cent, rectum 7 per cent, duodenum 5 per cent, retroperitoneal 4·5 per cent, colon 3 per cent. It should be noted, however, that these figures take account of all cases; for example, in their own series of 60 cases 30 were found incidentally at operation or autopsy, and no less than 25 of their 33 gastric cases were of this category. The surgeon might expect, therefore, to encounter rather fewer gastric cases than these figures would suggest.

Table I.—SMOOTH-MUSCLE TUMOURS OF STOMACH

Case No.	Age	Sex	Presenting Features	X-ray Findings	Tumour Site and Size	Histology	Follow-up
1	34	M.	Melæna	Ulcer—malignant	Lesser curvature, 3 cm. diam.	Sarcoma	Died 6 yr.—metastases
2	45	F.	Hæmatemesis; melæna	Filling defect	Pylorus, 7 cm. diam.	Myoma	Alive and well—9 yr.
3	70	F.	Melæna; epigastric pain	Filling defect	Near old G.E. stoma, 'large'	Sarcoma	Died—soon after
4	52	F	Melæna	Filling defect	Fundus, 'large'	Myoma	Died—'post-operative chest'
5	65	F.	Hæmatemesis	Ulcerated intramural tumour	Anterior wall, 9 × 4 cm.	Myoma	Alive and well—18 mth.
6	54	F.	Hæmatemesis	Filling defect	Lesser curvature, 6 × 4 × 4 cm.	Myoma	Alive and well—1 yr.
7	79	F.	Intestinal obstruction; coexisting Ca. coli	—	Lesser curvature, 4 cm.	Sarcoma	Alive and well—5 yr.

Cornell, Shehadi, and Sharnoff (1950) give the most recent account of the rare œsophageal leiomyomata and describe 5 cases of their own.

Cowdell (1950), in this country, has studied gastric leiomyomatous tumours, and reports on 10 cases occurring in the 1938–49 period at the Radcliffe Infirmary; 2 of these were incidental findings. Starr and Dockerty (1955) describe the leiomyomata of the small intestine, summarizing 230 cases in world literature and an additional 63 symptom-producing and 13 incidental cases of their own. It is worth noting that the incidental finding of a leiomyoma is much less likely in the small intestine, where quite a small tumour can be expected to produce symptoms.

Leiomyoma of the colon is distinctly rare. McKenzie, McDonald, and Waugh (1954) describe 13 cases from the Mayo Clinic and note a further 24 in the literature. The rectum is more frequently involved. Anderson, Dockerty, and Buie (1950) give an account of 20 cases and briefly survey the literature.

CASE REPORTS

1. Œsophagus.—The single case encountered was a male aged 42 years with a history of upper abdominal pain for five years suggestive of peptic ulceration. Barium examination revealed a filling defect in the œsophagus at 30 cm. (*Fig.* 718) and local enucleation with repair of the muscularis was carried out at thoracotomy. The patient made a good recovery and is alive and well seven years later. The tumour was an elongated and slightly irregular

mass, histologically a typical benign leiomyoma, the general nature of which will be described later.

2. Stomach (7 cases).—The main features of this group are given in *Table I*. Clinically there was nothing of specific value in diagnosis. The presenting symptoms were those due to bleeding, presumably from the ulcerated surface of the tumour. This was a feature in both benign and malignant tumours. Pain and tenderness in the epigastrium were inconstant findings and in no case was a mass palpated with certainty despite the relatively large size of tumours later found. On radiological examination the usual appearance was that of a large filling defect, although in 1 case the picture resembled that of a carcinomatous ulcer.

FIG. 718.—Filling defect in œsophagus, with smooth rounded contour. Barium swallow.

FIG. 719.—Gastric leiomyoma sectioned to show appearance of encapsulation.

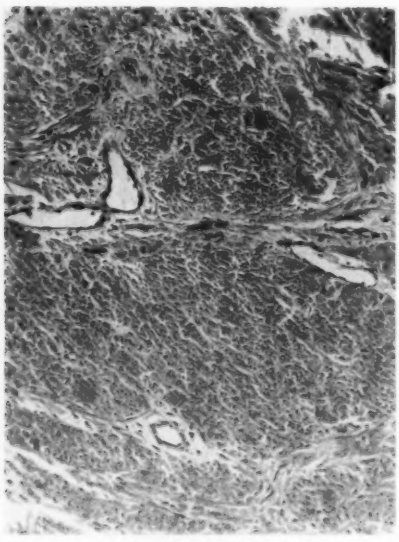

FIG. 720.—Benign gastric leiomyoma of typical appearance. H. and E. (× 56.)

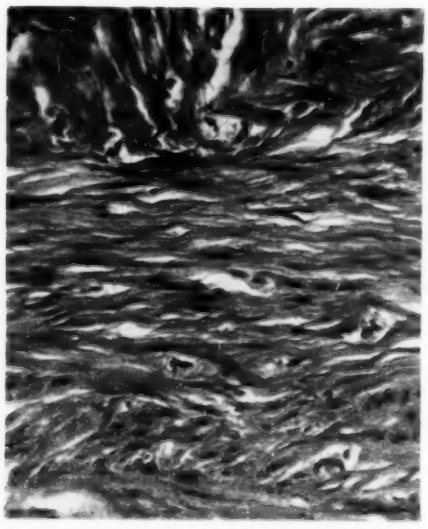

FIG. 721.—Benign gastric leiomyoma. H. and E. (× 208.)

In all cases the pre-operative diagnosis was that of a malignant gastric lesion and exploratory laparotomy was performed. The true nature of the tumour was usually apparent immediately in view of its large, smooth, rounded, and intragastric nature. Doubt might arise in a deeply ulcerated lesion or in a tumour still largely intramural and not bulging either into the

lumen or to the serosa. It is of interest to note that none of the cases presented as a mass arising from the muscle and growing to form a large extragastric tumour.

occurred in 2 instances. The sarcomatous tumours generally, but not invariably, felt softer and less circumscribed than the benign leiomyomata, which tended, in our view, to form a false capsule of

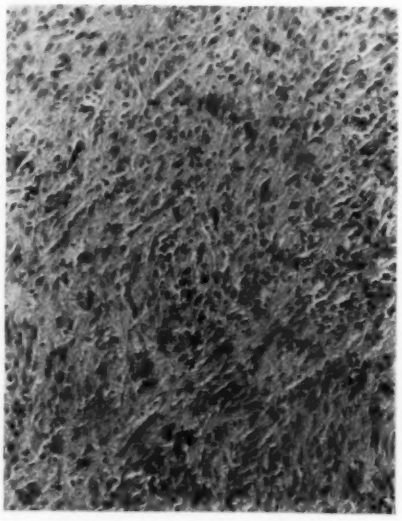

FIG. 722.—Benign gastric leiomyoma of more cellular pattern adjacent to an area of ulceration. H. and E. (× 80.)

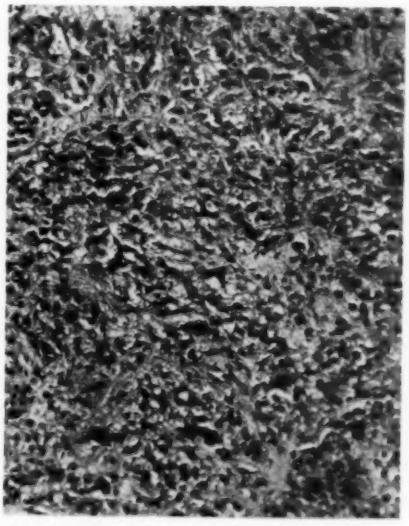

FIG. 723.—Gastric leiomyosarcoma showing grossly aberrant cellular pattern. H. and E. (× 96.)

Surgical treatment was local excision, or partial gastrectomy if there was any doubt about the exact status of the tumour or its degree of mischief.

Pathologically, the tumours were usually large and lobulated, bulging from the muscularis towards the lumen. They were covered by mucosa which was often superficially ulcerated. Deep ulceration compressed muscle from which they could be shelled out. An example of this is seen in *Fig.* 719.

Histologically the benign tumours show the well-known fasciculated leiomyomatous structure (*Figs.* 720, 721) with a slight connective-tissue stroma. Mitoses are few, but variation in cell size and nuclear hyperchromasia may give in some fields a false

Table II.—SMOOTH-MUSCLE TUMOURS OF SMALL INTESTINE

CASE NO.	AGE	SEX	PRESENTING FEATURES	EVIDENCE OF BLEEDING	TUMOUR SITE AND SIZE	HISTOLOGY	FOLLOW-UP
1	37	F.	Obstruction	No	Terminal ileum intramural, 7 cm. diam.	Myoma	Alive and well— 2 yr.
2	44	M.	Obstruction	No	Terminal ileum intramural, 5 cm. diam.	Sarcoma	Died—soon after
3	45	M.	Obstruction	Occult blood	Mid-jejunum polypoid, 2·5 cm. diam.	Myoma	Alive and well—10 yr.
4	73	F.	Obstruction	Melæna	? site subserous, 16 × 11 × 10 cm.	Myoma	Died—'post-operative chest'
5	61	M.	Abdominal pain	Melæna	Terminal ileum polypoid, 'small'	Myoma	Died after some years—cerebral hæmorrhage
6	33	F.	Pain and mass in R.I.F.	No	Ileum subserous, 5 cm. diam.	Sarcoma	Died—9 yr.—metastases
7	30	F.	Pain and mass in midline	No	? site subserous	Sarcoma	Died—soon after
8	37	F.	Huge abdominal mass	No	Terminal ileum subserous, 'huge'	Myoma	Alive and well—1 yr.
9	52	M.	Obstruction	?	? site polypoid, small	Myoma	Died—6 yr.—carcinoma of stomach
10	71	M.	Melæna	Melæna	Jejunum, 2 cm. diam.	Myoma	Alive and well—6 mth.
11	50	F.	Anæmia: abdominal pain	Melæna	Jejunum, 9 × 8 × 6 cm.	Sarcoma	Alive and well—6 mth.

impression of undue activity until it is realized that there is adjacent œdema, inflammation, or ulceration. An example of this is shown in *Fig.* 722. Such spurious aberration is, of course, common in the uterine leiomyoma. Sarcomatous tumours show a highly cellular and pleomorphic picture, with abundant and often abnormal mitoses and cellular aberration (*Fig.* 723). Only a part of the tumour may show fasciculation and so give a hint of the histogenesis of the neoplasm.

3. Small Intestine (11 cases).—The main features of this group are recorded in *Table II.* The

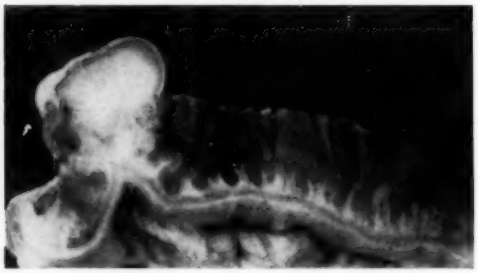

FIG. 724.—Polypoid benign leiomyoma of ileum.

presenting features are almost always those of intestinal obstruction, generally an acute obstruction superimposed on a history of vague abdominal pain of chronic partial obstructive nature. In 3 cases a mass was present as a symptom and in 2 of these pain of obstructive nature was also experienced. On clinical examination the findings were those of obstruction and need not be detailed.

In 5 cases there was evidence of bleeding from the tumour.

Laparotomy was performed in each case and, as with the gastric lesions, the true nature of the tumour was usually immediately apparent. Excision of the tumour or resection of the tumour-bearing segment of intestine was carried out without operative difficulty.

Pathologically, the tumours presented the same gross features as those in the stomach and could be intramural, with a serosal and intraluminal projection, or subserosal, or intraluminal and polypoid.

Intussusception occurred in 2 cases. The polypoid tumours, of which *Fig.* 724 is an example, are small and apparently produced symptoms early, while the intramural and subserous, especially the latter, could attain quite a large size before coming to clinical notice. The most common situation for the tumours seemed to be the lower ileum, but any part may be involved. No case occurred in the duodenum in this series. It was not possible with certainty to say on macroscopical evaluation whether the tumour was benign or malignant, although in most cases a fairly confident opinion could be given by reason of the relatively firm and demarcated nature of benign tumours.

Histologically the features are similar to those already described in gastric tumours, but there is one point of difference: cellular aberration, mitotic activity, and nuclear hyperchromasia are rather more frequent and occur even in quite small tumours which seem in every other respect, such as differentiation of muscle-fibres, fasciculation, and pseudo-encapsulation, to be benign. We interpret these features as of reactive nature rather than as manifestations of malignant change. They are certainly most noticeable in the polypoid tumours and we feel that peristaltic trauma and vascular changes are likely causes. If these aberrant foci are frequent it may be a matter of difficulty to decide upon the question of malignancy, and after taking all factors into consideration there may seem to be no alternative to reporting such cases as cellular leiomyomata of unpredictable behaviour.

4. Rectum (6 cases).—*Table III* gives the main facts in this group. In all cases the presenting features were indistinguishable from those of a rectal carcinoma, with rectal fullness, bleeding, and constipation. On rectal examination a mass was always felt, but it had no characteristic features and could be polypoid, smooth, and rounded, or hard and irregular. Biopsy at sigmoidoscopy revealed the true nature of the lesion.

In only one case was a radical excision of rectum carried out, and the tumour was found to be an ulcerated lesion with firm edges, the rectal wall being diffusely infiltrated by greyish tumour tissue. The macroscopical appearance was indistinguishable from a carcinoma. In the other cases, which occurred early in the series, local excision had been attempted and only fragments of tumour were available so that accurate morbid anatomical description was impossible.

Histologically 5 of the rectal tumours were frankly sarcomatous and presented no diagnostic

Table III.—SMOOTH-MUSCLE TUMOURS OF RECTUM

CASE NO.	AGE	SEX	PRESENTING FEATURES	RECTAL EXAMINATION	HISTOLOGY	FOLLOW-UP
1	61	F.	Bleeding; constipation	Large, soft mass anterior rectal wall	Myoma	Alive and well—10 yr.
2	81	M.	Anal mass	Firm ano-rectal mass	Sarcoma	Died—1 yr.
3	39	M.	Rectal fullness; bleeding	Polyp filling rectum—from left wall	Sarcoma	No follow-up
4	39	M.	Rectal pain	Large irregular mass anterior wall	Sarcoma	Died—1 yr.
5	31	M.	Bleeding, pain, and rectal fullness	Hard irregular mass anterior wall	Sarcoma	Died—1 yr.—metastases
6	79	F.	Bleeding; rectal fullness	Prolapsing mass in left wall	Sarcoma	Died soon after

difficulty. It is not possible in this series to say whether or not benign tumours might show aberrant foci such as are seen in tumours of the small intestine and thus occasionally cause diagnostic doubt. This difficulty was not encountered in the solitary benign lesion.

DISCUSSION

1. Clinical Features and Diagnosis.—The clinical features are simply those of a tumour mass projecting into a hollow viscus—namely, obstruction and hæmorrhage from the ulcerated surface, the size of the viscus determining which of these dominates the picture.

Diagnosis may be aided by radiology, especially in the œsophageal and gastric lesions, but even in these situations it is often difficult to differentiate leiomyomatous tumours from the common carcinomata. Moore (1927) gives a useful account of the criteria for radiological diagnosis. Frequently it will be only at laparotomy that the diagnosis is established. This almost invariably will be so in lesions of the small bowel when laparotomy is performed for obstruction. Persistent melæna with negative sigmoidoscopic and radiological findings in a patient with abdominal pain should suggest the possibility of a leiomyomatous tumour of small intestine and will certainly, in our view, merit laparotomy. We have found it impossible to differentiate clinically between sarcoma and carcinoma of the rectum, but because of the ease of biopsy, pre-operative diagnosis can always be established.

2. Pathology and Prognosis.—As has been already described, we note a tendency for benign tumours to become pseudo-encapsulated. We have not observed interdigitation of leiomyomatous fibres with surrounding muscle as described by Evans (1956) in his account of smooth-muscle tumours. We regard the macroscopical appearance of a leiomyomatous tumour as a useful but not infallible guide to its probable degree of activity. Histologically, in most instances there is little doubt, but, as we have shown, especially in cases affecting the small intestine, there is a small group of cases showing cellularity and aberration and it may be difficult to decide whether this is reactive or a sign of incipient malignancy. This is mentioned by most writers. Golden and Stout (1941) regarded in such cases the number of mitoses as an important criterion of malignancy, and stated that if two or more mitoses per high-power field are present one can feel fairly secure in predicting malignancy. But they admit the converse to be untrue, for some clinically malignant cases have shown relatively few mitoses. As Evans points out, "Unfortunately the paucity of mitotic figures in a leiomyoma does not augur a benign termination in all cases". In practice we allow more aberration of pattern in a congested polypoid tumour or a deeply ulcerated tumour than in an uncomplicated tumour before applying the label of malignancy. Willis (1953), speaking of the uterine leiomyomata, mentions that "a cellular leiomyomatous growth which arouses the pathologist's suspicion, but of the malignancy of which he feels uncertain, is usually benign". This is probably the most practical approach to the problem.

An attempt at careful follow-up of cases has been made to try to decide upon the likely behaviour of these tumours. In a series extending back over twenty years this is difficult as many cases have died out of hospital from other causes or without an accurate account of the final illness, and other cases are more recent and the follow-up period is short. We have found, however, despite these limitations, that the outcome of the cases has accorded well with the histological diagnosis, and in no case diagnosed as a benign leiomyoma has a recurrence, local or remote, been observed. It seems possible, therefore, that in those disturbingly cellular leiomyomata showing a degree of aberration but infrequent mitoses, especially in the polypoid small-intestinal or ulcerated gastric varieties, an optimistic histological report is warranted.

3. Treatment.—Surgery is clearly the treatment of choice for these tumours. Radiotherapy has occasionally been used, but has been dismissed by most authors as of no value. It was not attempted in our series, in which enucleation or some more radical form of excision has generally been carried out. It is justified, and indeed wise, to adopt a radical procedure unless at laparotomy the tumour is manifestly benign, when more local measures such as enucleation or local resection might be preferred.

SUMMARY

1. Twenty-five cases of leiomyomatous tumours of the alimentary tract are briefly described, 1 in œsophagus, 7 in stomach, 11 in small intestine, and 6 in rectum.

2. Three of the gastric, 4 of the intestinal, and 5 of the rectal cases were sarcomatous, the remainder being benign.

3. The clinical features are those of a tumour mass arising in the wall of a hollow viscus and causing either obstruction or hæmorrhage, depending upon the size of the viscus involved.

4. There is generally little difficulty in pathological diagnosis, but ulceration and congestion may produce aberration in cell pattern and arouse histological suspicion of malignancy. In such instances an optimistic report seems justified.

5. Surgical removal is the treatment of choice and a radical excision is preferred.

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EXPERIMENTAL SURGERY

A SURVEY OF THE ABSORBABILITY OF COMMERCIAL SURGICAL CATGUT

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THE object of this study was to survey the absorption characteristics of commercially available surgical catgut and to determine what variations exist among samples of catgut from different manufacturers but with a similar designation (e.g., 'plain', 'chromic', etc.). Previous surveys of this type in the U.S.A. (Jenkins, 1942; Jenkins and Hrdina, 1942 a, b; Jenkins, Hrdina, Owens, and Swisher, 1942; Wolff and Priestley, 1939) and in Great Britain (Holder, 1946) demonstrated that there was a very wide variation indeed in resistance to absorption among the different makes of similarly labelled catgut.

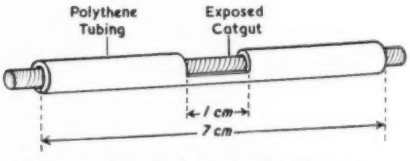

FIG. 725.—Catgut implant with polythene protector.

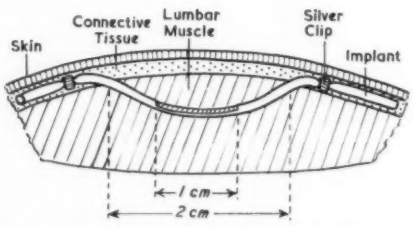

FIG. 727.—Section showing position of implant in the muscle.

Many changes have taken place in the suture industry since these surveys were done, and it was thought that it would be interesting to see if there was any move towards uniformity of absorption characteristics among different brands of catgut on the British market.

METHODS

The method of implantation in the lumbar muscles of rats used in these experiments was that developed by the author (Lawrie, 1955) and consists briefly of the following stages: (1) The sterile suture is cut into 3 in. lengths and threaded into a piece of polythene tubing with a 'window' cut in it as in *Fig.* 725. This method exposed a standard length of catgut to the tissues. (2) Two of these implants are inserted, under ether anæsthesia, through a large bore hypodermic needle into each lumbar muscle of a rat, as shown in *Figs.* 726–728, and anchored by

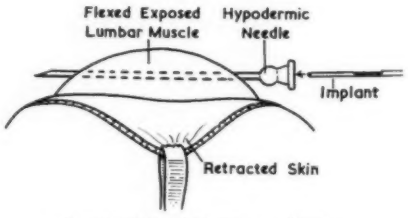

FIG. 726.—Method of insertion of implant.

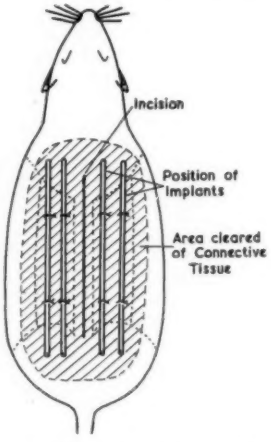

FIG. 728.—Diagram showing position of incision and implants.

means of silver hæmostasis clips. (3) After the required length of time the animal is killed, the implants are gently withdrawn, and the tensile strength of the suture is measured on an inclined

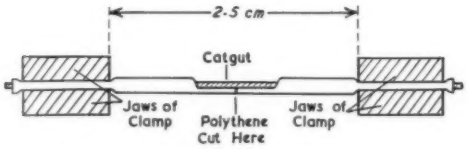

FIG. 729.—Method of testing for tensile strength.

plane tensiometer of the type specified in the *United States Pharmacopœia*, after cutting the polythene at the point shown in *Fig.* 729. (4) The absorption of plain catgut is compared with the same brand of chromic catgut. In *Fig.* 736 the various brands of plain catgut are compared and in *Fig.* 737 the various brands of chromic catgut.

Table II shows the approximate times for reduction to half the original strength and the approximate times for reduction to zero strength.

DISCUSSION

With the exception of brand E, the variation among the different makes of *plain* catgut was not very great. The variation, however, among the different makes of *chromic* catgut was very wide, the 'half-strength time' varying from about 6 days for brand C to about 22 days for brands A and E.

Another rather disturbing fact is that there is virtually no difference in the absorption properties of plain and chromic catgut of brands C, D, and E.

Table I.—AVERAGE TENSILE STRENGTH (lb.)

TIME (Days)	BRAND A		BRAND B		BRAND C		BRAND D		BRAND E		BRAND F	
	Plain	Chromic	Plain	Chromic	Plain	Chromic	Plain	Chromic	Plain	Chromic	Plain	Chromic
1	3·02	3·76	2·74	3·57	2·58	3·13	2·70	2·73	3·90	3·85	2·47	2·94
2	3·12	—	1·60	—	2·57	—	1·73	—	4·60	—	2·03	—
3	2·70	2·74	1·58	3·25	1·70	2·05	1·52	1·93	3·42	4·13	1·17	2·44
5	1·92	3·59	0·66	2·79	2·34	1·57	1·71	1·48	3·92	3·26	1·31	2·62
7	1·06	3·38	0·29	2·90	2·58	1·47	1·70	1·01	3·47	3·87	1·09	1·90
10	1·27	4·74	0·20	2·97	1·11	1·48	0·70	0·78	3·03	3·07	0·18	1·43
15	1·14	2·95	0·02	1·50	0·65	0·86	0·71	0·50	2·36	2·79	0·55	0·73
20	0·41	1·86	0	0·49	0·90	0·04	0·09	0·10	1·57	2·60	0	0·11
25	0	2·18	0	0·13	—	0·06	0·05	0·02	1·27	1·32	0	0
30	0·05	0·97	—	0·09	—	0	0	0·02	1·85	1·29	—	0·02
35	—	—	—	0	—	0	—	0	—	2·82	—	0·01
40	0	0·54	—	—	—	—	—	—	1·58	0·46	—	—
50	0·01	0	—	—	—	—	—	—	—	1·06	—	—
60	—	0·67	—	—	—	—	—	—	—	—	—	—

characteristics are demonstrated by plotting the tensile strength against the time in the tissues, using the average tensile strength of 20 implants (5 rats) for each point on the graph.

Two values may then be deduced from the graph:—

a. The time taken for the tensile strength to be reduced to zero.

b. The time taken for the tensile strength to be reduced to half its original value.

The latter figure is probably more significant clinically because a fine suture would be of little value in holding the edges of a wound together after it had been reduced beyond half its original strength.

Samples of Catgut.—Samples of catgut from six different British manufacturers were purchased on the open market and were of two types: 'plain' and 'chromic'. The description of the latter varied among the different manufacturers (e.g., 'medium chromic', 'medium hard chromic', '20 day', etc.), but in this paper all will be referred to as 'chromic'. In all except brands A and C, where only one batch was available, the results are the average of two different batches.

EXPERIMENTAL RESULTS

Table I shows the average tensile strengths obtained after various times in rat lumbar muscle, and the graphs, *Figs.* 730–735, show the average tensile strength plotted against time, and each brand

Clearly some standardization is needed, but no such standards for absorption properties are yet to be found in the *British Pharmacopœia* or in the *United States Pharmacopœia*. The latter suggests four categories, viz., Type A or Plain, Type B or Mild Chromic, Type C or Medium Chromic, and Type D or Hard Chromic, but no guidance is given as to the

Table II.—TIMES FOR REDUCTION TO HALF STRENGTH AND ZERO STRENGTH

BRAND	PLAIN		CHROMIC	
	Half Strength	Zero Strength	Half Strength	Zero Strength
	days	days	days	days
A	8	25	22	50–60
B	3	15	14	30–35
C	9	25–30	6	20–30
D	7	25–30	7	25–30
E	18	60+	22	60+
F	4	20	10	25–30

actual absorption time expected from these four types.

The designation '20 day', '30 day', etc., catgut appears at first sight to be rather more explicit, but such a description raises the question: "What does the surgeon expect to have happened after the 20 or 30 days have elapsed?" Should the catgut be reduced to zero strength, to sufficient strength to hold

the wound? (if so, what sort of wound?), or should the catgut be completely absorbed histologically? In actual fact, especially when chromic catgut is used, histological absorption is seldom complete until weeks and sometimes months after the tensile strength has been reduced to zero.

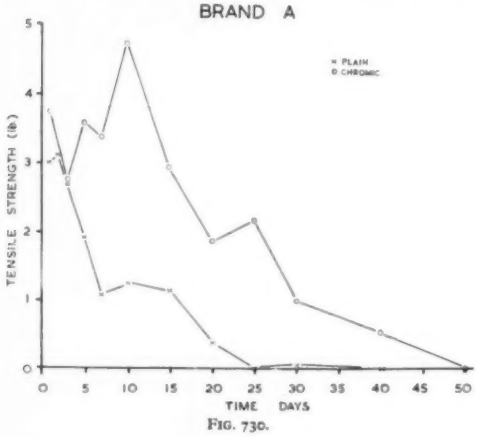

FIG. 730.

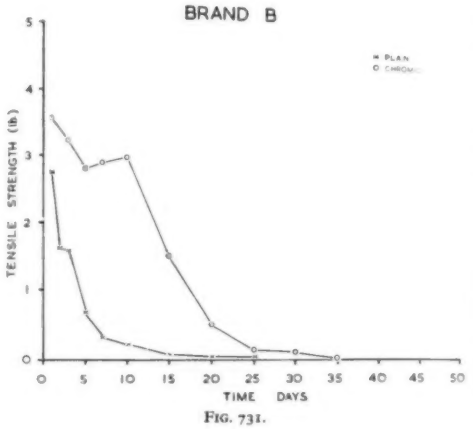

FIG. 731.

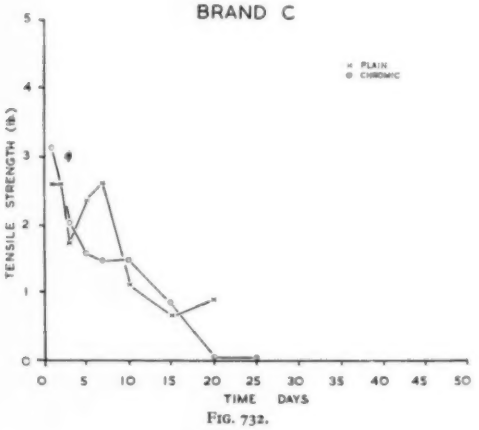

FIG. 732.

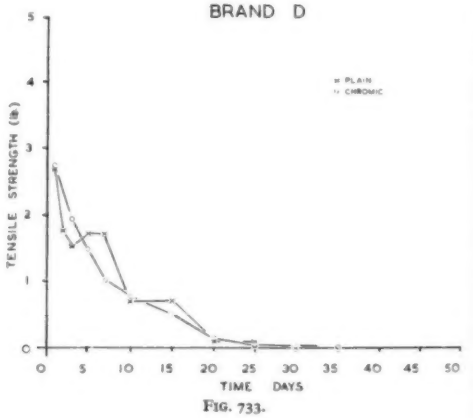

FIG. 733.

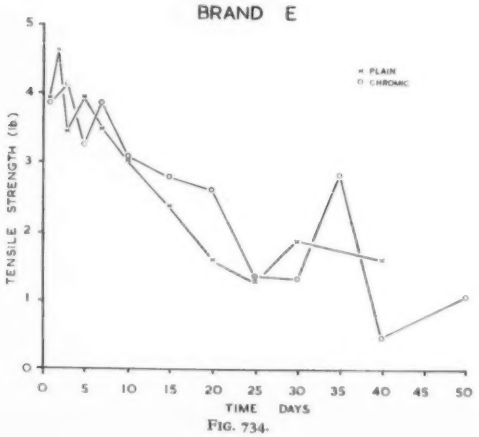

FIG. 734.

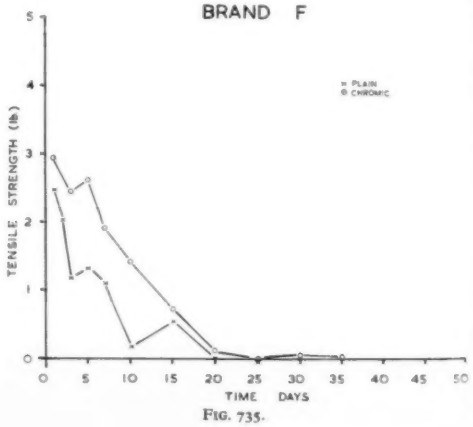

FIG. 735.

There is no clear indication from the surgical profession as to what absorption characteristics are desirable in surgical catgut and, in spite of the variations of these brands of catgut, it is clear that

SUMMARY

A survey of the absorption properties of plain and chromic catgut from six different British manufacturers has been made.

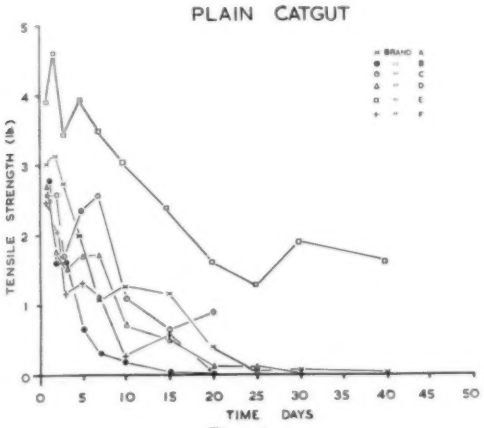

FIG. 736.

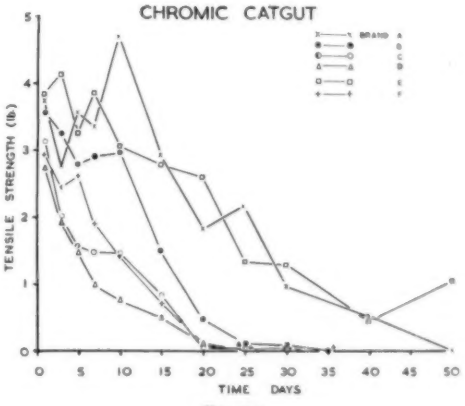

FIG. 737.

all of them must give an appreciable degree of satisfaction to the surgeon and that the resistance to absorption of all of them is adequate for normal surgical purposes. Indeed, it is probably true to say that the rate of healing in a normal surgical wound is fast enough to allow even the 'plainest of plain' catgut to be used with safety, and it is only when wound healing is delayed that a more durable suture is necessary.

It is evidently highly desirable that there should be some official pharmacopœial standards for the principal property of catgut that commends its use as a suture—namely, its absorbability. It is suggested that the method outlined above might form the basis of an absorption test for inclusion in the *British Pharmacopœia*, but this could only be introduced after prolonged clinical trials of suitably standardized material. Suitable limits for the 'half-strength' time in rat muscle for such clinical material might be: Type A—3 to 7 days; Type B—5 to 10 days; Type C—15 to 25 days; Type D—25 to 35 days.

Of these four types perhaps only two might ultimately be deemed adequate.

Wide variation in the absorption rates of these samples of catgut has been found, especially in the chromic material, and it is suggested that the time has come when official standards for absorption rate should be included in the *British Pharmacopœia* and tentative standards for clinical evaluation have been suggested.

Acknowledgements.—My thanks are due to the Directors of Ethicon Ltd., and to the Royal College of Surgeons of England for permission to publish this work.

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THE ABSORPTION OF SURGICAL CATGUT

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WHEN Lister's antiseptic methods allowed healing by first intention, it became possible to use sutures which could be absorbed by the tissues. Catgut, a material long in use, was the obvious choice. Catgut, like leather, is collagen, and Lister experimented with many tanning agents, finally concluding that chrome salts were the most suitable (Lister, 1908).

To-day, both plain and chromic catgut are widely used by surgeons. There is great variation in the absorption rates of different brands (Jenkins, Hrdina, Owens, and Swisher, 1942) and it has been suggested that plain catgut fails to hold long enough for the healing wound to become secure (Bates, 1939).

To compare the effects in the tissues of plain and medium chromic catgut, we have implanted these sutures in the lumbar muscles of the rat. In one series we have examined them histologically at different intervals of time. In another we have measured the tensile strength of these sutures at the same intervals after implantation.

MATERIALS AND METHODS

Wistar albino rats of average weight, 160 g., were fed on standard rat diet 41B with free access to food and water. They were housed in a room the temperature of which never fell below 60° F. The rats were anæsthetized with ether, and under aseptic conditions a midline dorsal incision was made in the skin. For histological examination the implantations were made twice on each side through the exposed lumbar muscle with a 45-mm. curved intestinal needle. To mark the point of insertion of the catgut and to anchor its free ends, a suture of 5/0 black braided silk was inserted with a 16-mm. curved triangular cutting needle. A control experiment was performed by merely passing the 45-mm. intestinal needle through the lumbar muscle without leaving a catgut implant. The point of insertion of the needle was again marked with silk. A total of 163 rats were used. After varying intervals, the animals were killed with chloroform and the lumbar muscles were excised and fixed in 10 per cent formol saline. Blocks were embedded in paraffin and transverse sections were cut at 6 μ and stained with hæmatoxylin and eosin.

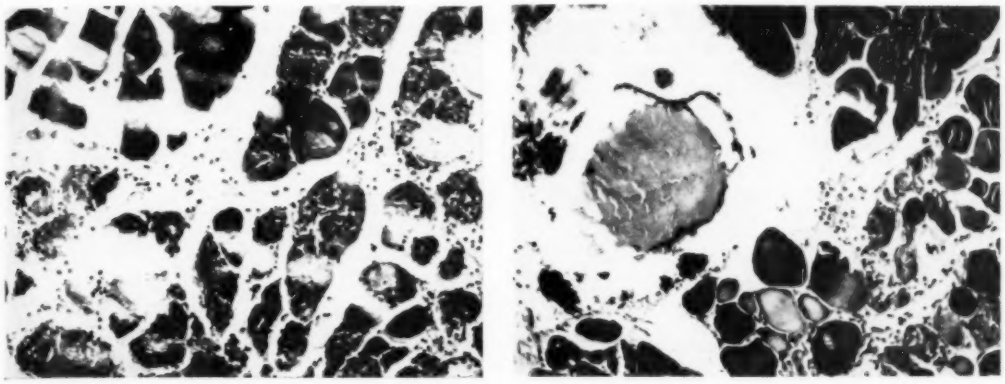
For examination of the tensile strength, the catgut was first passed through a polythene tube having a window 1 cm. long cut out of one side. Polythene tubes with catgut were then implanted in the lumbar muscles, two on each side of the rat. Both ends of the tubes were anchored by silver hæmostasis clips where they entered and left the muscle. A total of 170 rats were used. The rats were killed with chloroform and the silver clips were removed. The implants were gently withdrawn and tested immediately on an inclined plane tensiometer as specified in the *United States Pharmacopœia*. Lawrie (1955) gives the method in greater detail.

RESULTS

Histology.—When the muscle was merely injured by the needle without implanting catgut, a mild inflammatory reaction resulted with the removal of those muscle cells which were actually damaged by the passage of the needle. Material taken three hours after the operation showed a little fresh hæmorrhage with a few polymorphonuclear leucocytes. In six hours polymorphs were present over a wider area, but they were sparse and it was difficult to identify the muscle cells which had been damaged. By nine hours damaged muscle cells showed obvious autolytic changes and were invaded by polymorphs. There was a little œdema between muscle-bundles and the first macrophages appeared. Œdema and polymorphs reached a maximum by twelve hours and damaged muscle cells were beginning to be removed by the leucocytes, which were now heavily invading them (*Fig.* 738 A). Macrophages were becoming more numerous. In eighteen hours the œdema had subsided a little and the muscle cells containing polymorphs looked very 'moth-eaten'. By twenty-four hours most of the damaged muscle had been removed. Polymorphs by this time were scanty, muscle had been completely removed, and œdema had disappeared. There was a little sarcolemmal proliferation and a number of macrophages surrounded healthy muscle. By the fourth day there were only a few macrophages left and by the sixth day there was complete return to normal.

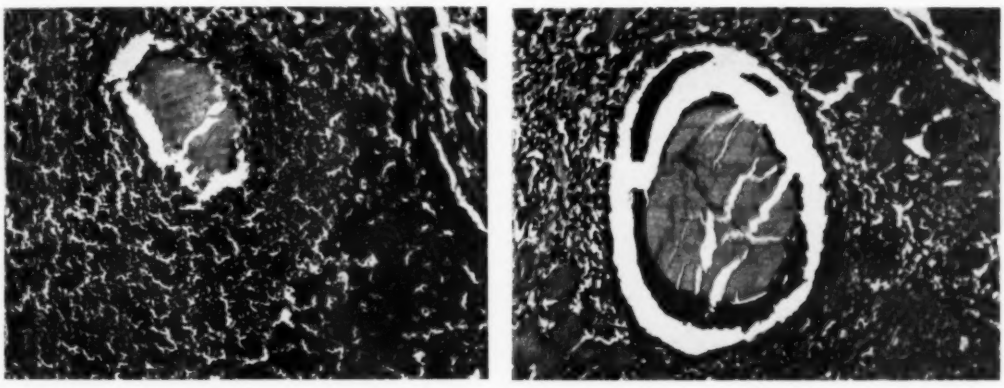
Plain Catgut.—Implants of both 3/0 and 5/0 plain catgut behaved similarly. At first the muscle surrounding the suture showed similar changes to those in the muscle damaged by passage of a needle alone. Œdema and polymorphonuclear infiltration reached their height at 12–18 hours, and in two days all the dead muscle cells had been removed and the œdema had subsided. By the third day there was a thin wall of granulation tissue sharply separating healthy muscle from the channel containing the suture.

A little fibrinous exudate first appeared around the suture at nine hours. By twelve hours there was more exudate which contained stringy material staining blue with hæmatoxylin. After eighteen hours the exudate contained many polymorphonuclear cells, some of which had degenerated, and it was the nuclear debris of these cells which formed the blue stringy material. From two days onward attack on the suture itself was evident. Its diameter had been reduced by about one-third and in some places it was crumbling. At the third day the suture was fragmented and its area of cross-section was halved. It was surrounded by an abscess about six times its diameter. The pus in this abscess was divided into two zones, an inner one of polymorphs and an outer one of cell debris and blue-staining material. The muscle was compressed



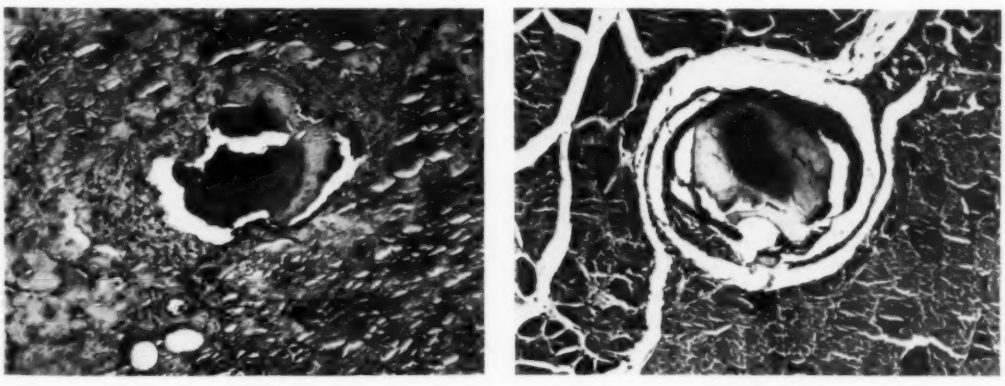
A B

FIG. 738.—A, Muscle twelve hours after injury by needle without implantation of suture. Damaged muscle cells are autolysed and invaded by leucocytes. B, Medium chromic catgut twelve hours after implantation. The state of the damaged muscle is the same as in A. There is a small amount of blue stringy material around the suture. H. and E. (× 120.)



A B

FIG. 739.—Catgut three days after implantation. A, Plain catgut. The suture is reduced by half. The large abscess is divided into an inner zone of polymorphs and an outer zone of cell debris and blue stringy material. B, Medium chromic catgut. The suture has not been absorbed. There is a slight polymorph reaction with some blue stringy material. The cracks in the gut are an artefact. H. and E. (× 120.)

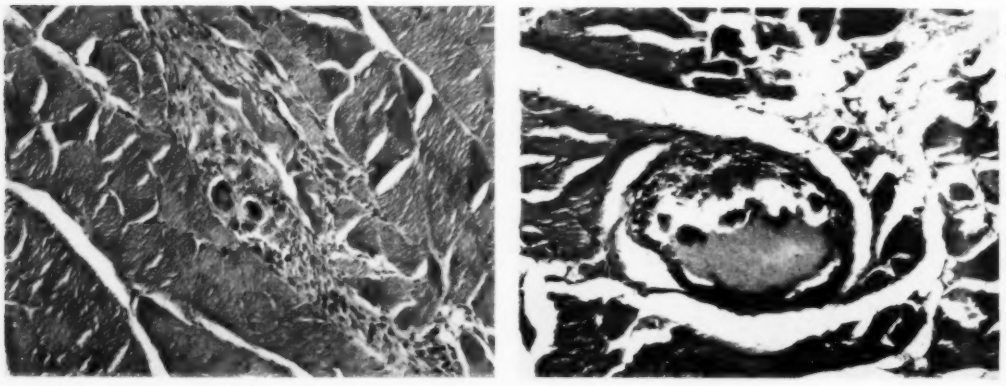


A B

FIG. 740.—Catgut ten days after implantation. A, Plain catgut. A suture reduced by more than half with a giant-cell reaction. The abscess has completely absorbed. B, Medium chromic catgut. A single giant cell is removing a 'bite' of suture, but has become displaced by the microtome. H. and E. (× 120.)

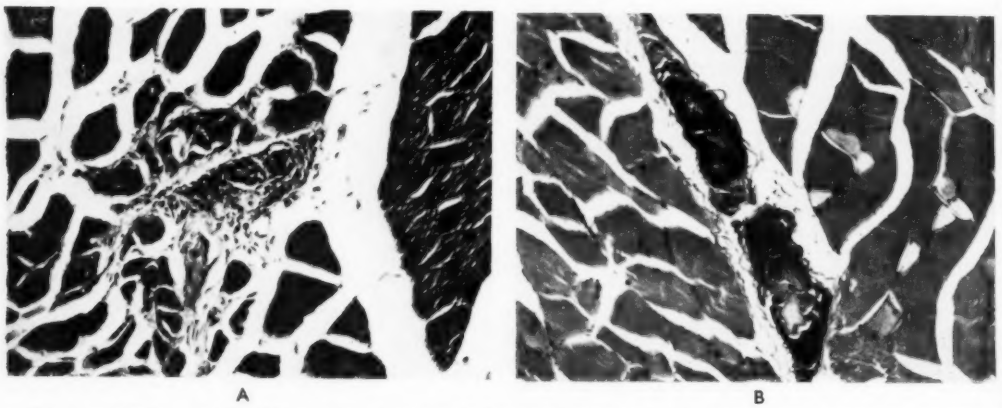
by the abscess and separated from it by a sleeve of compact granulation tissue (*Fig.* 739 A). At first it was thought that this abscess was caused by infection, but careful repetition of the experiment dispelled this idea. The abscess was slowly absorbed until on the seventh day there was no pus or blue debris. The lesion by this time had contracted to three times the diameter of the suture and consisted of granulation

After twelve hours exudate containing blue stringy material was seen around the suture (*Fig.* 738 B). This reached a maximum in three days, when the lesion was about twice the diameter of the suture and consisted only of dead polymorphs and blue stringy nuclear debris (*Fig.* 739 B). The brisk pyogenic reaction to plain catgut was absent and the suture itself appeared to be unaffected. By five days



A B

FIG. 741.—Catgut twenty-five days after implantation. A, Plain catgut. Fragments of suture persist in giant cells. B, Medium chromic catgut. Only half the suture has been removed and there are many giant cells. H. and E. (× 120.)



A B

FIG. 742.—A, Plain catgut after thirty-five days shows a small cluster of giant cells only. B, Medium chromic catgut after fifty-five days. Fragments of suture persist in giant cells. H. and E. (× 120.)

tissue containing numerous giant cells. The cross-section of the suture was only about half its former area and had 'bites' taken out of its circumference by giant cells (*Fig.* 740 A). The granulation tissue then progressively diminished in amount, foreign body giant cells removing more and more of the suture. Fragments of suture persisted until the twenty-fifth day (*Fig.* 741 A). On the thirty-fifth day only a minute giant-cell cluster was left (*Fig.* 742 A).

Medium Chromic Catgut.—The appearances produced by both 3/0 and 5/0 catgut were similar. The early inflammatory changes with removal of damaged muscle cells followed the usual pattern, and were complete in two days.

there was a narrow wall of granulations with no surrounding inflammation. All the exudate had disappeared by seven days. The first giant cell was seen on the tenth day, taking a 'bite' out of the circumference of the suture (*Fig.* 740 B). The number of giant cells steadily increased, so that on the seventeenth day most of the surface of the suture had been attacked. On the twenty-fifth day about half the area of cross-section of the gut had been removed (*Fig.* 741 B), and by the fifty-fifth day only traces remained (*Fig.* 742 B). Absorption was complete by the sixtieth day, and all that remained at seventy days was a tiny cluster of giant cells. At ninety days only a single giant cell was left.

Tensile Strength.—The tensile strength in pounds of the samples of catgut used at varying time intervals in the rat's muscle are given in *Tables I–IV*.

DISCUSSION

There are two phases in the weakening and absorption of catgut in the tissues (Jenkins and others, 1942).

Table I.—TENSILE STRENGTH OF 3/0 PLAIN CATGUT

DAYS SINCE IMPLANTATION	NUMBER OF TESTS	AVERAGE TENSILE STRENGTH (lb.)	NUMBER OF TESTS WHERE TENSILE STRENGTH WAS NIL
Dry, unimplanted	20	5·85	0
1	20	2·15	0
2	20	1·16	1
3	19	0·54	6
5	20	0·79	4
7	20	0·30	11
10	16	0·43	6
15	14	0·17	9
20	20	nil	20

The first phase of digestion by polymorphonuclear leucocytes begins about twelve hours after implantation and reaches a maximum in three days. The second phase of gradual removal by foreign body giant cells and macrophages does not begin until the seventh or tenth day and persists until the suture has been completely absorbed. The most striking feature of our results is the difference between plain and medium chromic catgut in the first phase (*Fig.* 739). Plain catgut evokes a brisk pyogenic reaction which at three days produces an abscess of considerable size. The proteolytic enzymes in this pus attack the suture and its tensile strength diminishes rapidly, so that by the third day a few of the sutures have lost their strength completely. The pyogenic reaction to medium chromic catgut is much less. There is a zone of dead polymorphs and blue debris around the suture at three days but the gut itself appears in the section to be unaffected and indeed it does not lose greatly in tensile strength until about the tenth day, by which time it is being attacked by giant cells (*Fig.* 740). Clearly, from these results, only medium chromic sutures can be relied on to last until healing of the wound is established.

Table III.—TENSILE STRENGTH OF 3/0 MEDIUM CHROMIC CATGUT

DAYS SINCE IMPLANTATION	NUMBER OF TESTS	AVERAGE TENSILE STRENGTH (lb.)	NUMBER OF TESTS WHERE TENSILE STRENGTH WAS NIL
Dry, unimplanted	20	4·65	0
1	20	4·48	0
3	20	4·03	0
5	20	3·49	0
7	20	3·02	0
10	20	2·04	0
15	20	2·53	0
20	20	1·30	3
30	20	0·37	12
40	20	nil	20

Howes and Harvey (1930) and Burke (1940) both stated that medium chromic catgut produced more reaction in the tissues than plain gut. We cannot agree. Bates (1939) concluded that plain catgut in all sizes excited a prompt, violent, exudative foreign-body reaction and failed to hold throughout the time wound support was needed. He favoured the use of fine chromic gut, which functioned longer than the larger sizes in his experience. Mahoney (1943) and Pickrell and Clay (1944) also found that the smaller sizes were less rapidly weakened than the larger. In our experiments we have only used fine catgut and there is no great difference between 3/0 and 5/0 in the rates at which they lose tensile strength. However, there is a great difference between plain and medium chromic catgut, the former losing strength in three days, the latter remaining strong for ten or fifteen days, by which time healing is complete.

Table II.—TENSILE STRENGTH OF 5/0 PLAIN CATGUT

DAYS SINCE IMPLANTATION	NUMBER OF TESTS	AVERAGE TENSILE STRENGTH (lb.)	NUMBER OF TESTS WHERE TENSILE STRENGTH WAS NIL
Dry, unimplanted	20	1·50	0
1	20	1·10	0
2	18	0·75	0
3	20	0·64	2
5	20	0·24	3
7	20	0·24	8
10	20	0·18	10
15	20	0·04	15

Table IV.—TENSILE STRENGTH OF 5/0 MEDIUM CHROMIC CATGUT

DAYS SINCE IMPLANTATION	NUMBER OF TESTS	AVERAGE TENSILE STRENGTH (lb.)	NUMBER OF TESTS WHERE TENSILE STRENGTH WAS NIL
Dry, unimplanted	20	1·52	0
1	20	0·95	0
2	20	1·10	0
3	20	0·85	0
5	20	1·01	0
7	20	0·81	0
10	16	0·88	0
15	20	0·53	2
20	18	0·68	0
25	19	0·22	5
30	20	0·20	11

SUMMARY

Fine catgut was implanted into the lumbar muscle of rats which were afterwards killed at intervals between three hours and ninety days. The tensile strength of the catgut was then measured and material was taken for histological examination.

Plain catgut evoked a severe sterile pyogenic reaction in the tissues in three days which led to a rapid loss of tensile strength. Medium chromic catgut produced a very slight inflammatory reaction at first and was not substantially weakened until foreign-body giant cells began attacking it ten days after implantation.

It is concluded that plain catgut is unreliable as a suture material for longer than two days, whereas medium chromic catgut always retains adequate strength long enough for firm union of wound edges to take place in the absence of infection.

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of Surgeons of England for permission to publish this work; and to Professor G. J. Cunningham, Department of Pathology, Royal College of Surgeons of England, for his help and advice; and to Mr. A. L. E. Barron, of the same department, for the photomicrographs.

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THE STRENGTH AND HISTOLOGY OF SKIN WOUNDS IN LATHYRISM

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IT has been recognized since biblical times that excessive ingestion of certain lathyrus pulses may result in a syndrome characterized by neurological disturbances. Recently, it has been discovered that the feeding to experimental animals of diets rich in the seeds of *Lathyrus odoratus*, the sweet pea, may result in abnormalities in mesenchymal tissues, particularly of the skeleton and blood-vessels (Geiger, Steenbock, and Parsons, 1933; Ponseti and Shepard, 1954). Selye (1957) has reviewed extensively the literature on both human and experimental lathyrism. The pathogenic factor in the seeds of *Lathyrus odoratus* is β-aminopropio: nitrile (Schilling and Strong, 1955), but its mode of action on mesenchymal tissues, for example on collagen, is obscure.

As rat skin is particularly rich in collagen, the effect of lathyrism on the formation of collagen can conveniently be investigated in healing skin wounds. In the present experiments wound tensile strength has been measured, and correlated with the histological appearance, at intervals up to 21 days after wounding. The influence of thyroxine on wound healing in lathyrism has also been investigated, this hormone having a pronounced inhibitory effect on the development of osteolathyrism (Selye, 1957).

Table I.—THE DIETARY TREATMENT AND THE PERIOD BETWEEN OPERATION AND KILLING OF THE GROUPS OF RATS USED

SURVIVAL PERIOD	GREEN-PEA DIET	SWEET-PEA DIET	SWEET-PEA DIET PLUS THYROXINE
1 week	15 rats	15 rats	
2 weeks	15	15	
3 weeks	14	13	
2 weeks		15	12

METHODS OF STUDY

Young adult white rats were used. A control group was fed *ad lib.* on a diet consisting half of casein, starch, sucrose, vitamins, and minerals (Hubbel, Mendel, and Wakeman, 1937), and half of green-pea meal. A second group was fed the same diet except that sweet-pea meal was substituted for the green-pea meal. On the day after the animals were put on the diet, a 2-cm. transverse skin wound was made on either side of the vertebral column at the level of the thoracolumbar junction, intraperitoneal nembutal anæsthesia and an aseptic technique being used. The wounds were closed in an identical manner with four silk sutures which were removed one week later. These animals were killed at one, two, and three weeks after operation. A further 27 animals were fed on the 50 per cent sweet-pea diet, and 12 of these were given sodium thyroxinate (B.D.H.) in a daily dose of 0·5 mg. subcutaneously. All were operated on as described above, and were killed two weeks later (*Table I*).

Table II.—A COMPARISON OF THE MEAN TENSILE STRENGTH OF 1 CM. OF HEALING WOUND IN THE NORMAL AND LATHYRITIC ANIMALS

SURVIVAL PERIOD	MEAN TENSILE STRENGTH IN G.		RATIO	P
	Green-pea Diet	Sweet-pea Diet		
1 week	335	228	1·5 : 1	<0·05
2 weeks	735	340	2 : 1	<0·01
3 weeks	789	258	3 : 1	<0·01

Table III.—THE EFFECT OF THYROXINE ON WOUND TENSILE STRENGTH IN LATHYRITIC RATS FOURTEEN DAYS AFTER OPERATION

	SWEET-PEA DIET (15 rats)	SWEET-PEA DIET PLUS THYROXINE (12 rats)
Mean tensile strength in g.	651	698
	No significant difference	

Immediately after killing, a strip of skin 1 cm. wide was cut at right angles to the wound with a twin-bladed scalpel, the wound crossing the centre of the strip. The tensile strength of the wound was then measured by applying a distracting force to the two ends of the strip (Douglas, 1957), the result being

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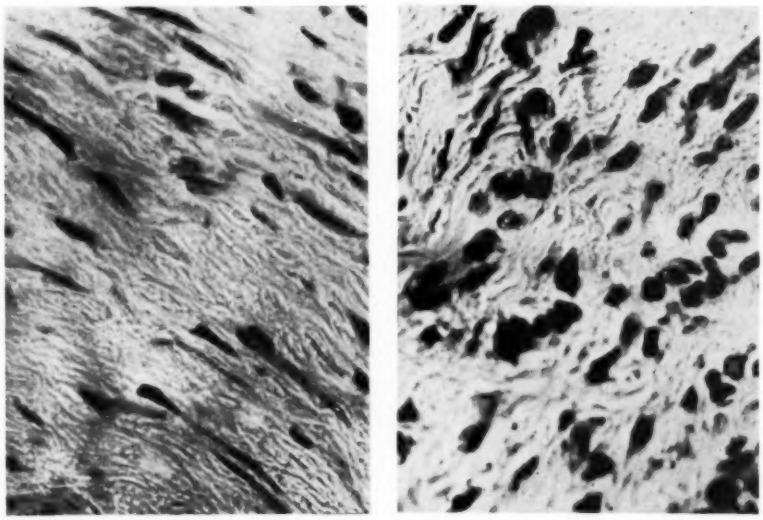
expressed in grammes. Two wounds were tested in each animal.

Portions of skin wounds with adjoining skin from approximately half of each group of animals were fixed in 10 per cent neutral formalin solution at 0 to 4° C. Frozen sections were cut, approximately 15 μ in thickness, and stained for alkaline phosphatase with 'Fast Blue B' salt (I.C.I.) for azo coupling (Pearse, 1953). Further tissue was embedded in paraffin wax, and 6-μ sections cut, some sections being stained with hæmalum and eosin to demonstrate collagen fibres, and others impregnated with silver solution to demonstrate reticular fibres, and resorcin-fuchsin for elastic fibres (Carleton and Drury, 1957). Sections were also processed by the periodic acid Schiff (PAS) technique (Hotchkiss, 1948). Tissues from each experimental and corresponding control group of animals were always stained together by each technique.



A B

FIG. 743.—Photomicrographs of fibrous tissue from wounds of (A) control and (B) lathyritic rats three weeks after operation. A, The nuclei of fibroblasts are spindle-shaped, and regular in size and arrangement. B, The fibroblast nuclei are larger, but vary considerably in size and shape. There is a subnormal amount of collagen. (× 560.)

RESULTS

Tensile Strength Studies.—The results of the tensile strength tests are set out in *Table II*. Wounds in the green pea (control) animals are stronger than wounds in the animals on the lathyritic diet at each of the three survival periods, though the difference is only on the border line of significance at one week after wounding. The addition of thyroxine had no significant effect on the development of wound tensile strength in lathyritic animals (*Table III*), the mean tensile strength of the wounds of the group of rats on the sweet-pea diet plus thyroxine being similar to that of rats on the sweet-pea diet without thyroxine. The rats receiving thyroxine lost 25 per cent of their body-weight during the course of the experiment.

Histology of the Wounds.—In skin wounds of animals given *Lathyrus odoratus* for one week reticular fibres are finer, and laid down in larger meshes, than those in skin wounds from control animals. There is no appreciable difference between elastic fibre distribution or concentration in wounds of control and experimental animals. Collagen fibres are distributed similarly in the dermis surrounding the wounds in all groups of animals. Fibroblasts are more irregular in shape, and larger, in wounds of lathyritic animals than in those of controls (*Fig.* 743), and their cytoplasm is generally more basophilic. Owing probably to the greater cellularity of wounds from experimental animals, there is less collagen in these wounds. These differences are most marked in animals given the experimental diet for three weeks. The mode of distribution of collagen is similar in wounds of control and experimental animals.

PAS-positive material is present in approximately equal amounts in control and experimental material. Phosphatase concentration seems to be slightly raised in the latter, but differences in concentration of this enzyme are small. Wounds of animals on the sweet-pea diet plus thyroxine are indistinguishable from those of animals on this diet without the hormone.

DISCUSSION

In normal wound healing the gradual increase in tensile strength is accompanied by a simultaneous formation of new collagen (Dunphy and Udupa, 1955). The greatly reduced strength of the wounds of lathyritic rats could therefore be due to impaired collagen formation, either a delay in normal formation or formation of an abnormal type. The calculation of tensile strength in these experiments, however, does

not take into account variation in thickness of the skin, so that another reason for the lower tensile strength might be the skin as a whole becoming thinner in lathyritic animals, the wounds, therefore, being weaker even if collagen is normal. The skin of the lathyritic animals at two weeks was, in fact, 12 per cent thinner than that of control animals. Dasler (1954) also noted this thinning of the skin in lathyrism, and we have shown elsewhere (Bell, Dunbar, Gillespie, Iball, and Oliver, 1959) that the tensile strength of unwounded skin is lower in lathyritic than in normal rats. While thinning of the skin as a whole is thus undoubtedly in part responsible for the reduced tensile strength in these experiments, there was also good correlation between subnormal tensile strength and histological evidence of impaired collagen formation.

The staining properties of fibres in skin wounds of lathyritic animals were similar to those in control wounds in our experiments. There was no increase in staining with resorcin-fuchsin. Since degeneration of a small amount of collagen produces elastin-like material (Keech and Reed, 1957) which has an increased affinity for resorcin-fuchsin (Tunbridge, Tattersall, Hall, Astbury, and Reed, 1952), it is unlikely that degeneration of collagen had occurred in the connective-tissue either of the wounds or of the surrounding dermis in the lathyritic animals. The reduced tensile strength in these wounds is, therefore, probably not due to degeneration of collagen. It may in fact be accounted for largely by a persisting cellularity of the healing tissue, with a consequent reduction in the amount of collagen laid down. It is interesting to note that Yeager and Hamre (1957) observed fibroplasia in the periosteum at muscle insertions in the femora of lathyritic rats. Their finding that the nuclei of these fibroblasts were larger than normal, and that their cytoplasm showed an increased basophilia, is similar to ours in this condition. Enzinger and Warner (1957) also found an increased cellularity in healing abdominal muscle wounds of lathyritic rats, in association with decreased tensile strength. In contrast, Krikos and Orbison (1958) reported a diminution in the number of fibroblasts in most parts of healing wounds in similar experiments.

Ponseti and Shepard (1954) consider that the lesions of mesodermal tissues in animals given *Lathyrus odoratus* seeds may be due to a defect in the mucopolysaccharide present. Aortic lesions in lathyritic rats were stated by Churchill, Gelfant, Lalich, and Angevine (1955) to be accompanied by an increase in PAS-positive material. Our finding of a normal content and distribution of polysaccharide in skin wounds in lathyrism is in contrast with this.

The experiments also yield some data as to the rate of recovery of tensile strength in skin wounds of normally fed rats. There is a large increase in wound strength between one and two weeks ($P = < 0{\cdot}01$) in the rats on the green-pea diet, but there is no significant further increase in strength between two and three weeks. Recovery of strength of skin wounds in the rat is thus largely complete by the end of two weeks, and any further increase in strength is small. The position in regard to the rate of wound healing in the lathyritic rats is somewhat obscure. There is an apparent increase in tensile strength between one and two weeks, but this increase is not significant. Wound strength appears to fall between two and three weeks, but the difference again is not significant.

SUMMARY

1. The tensile strength and histology of healing skin wounds in normal and lathyritic rats was compared one, two, and three weeks after wounding.

2. Wounds of lathyritic rats showed a greatly reduced tensile strength at all stages.

3. There was greater cellularity, and less collagen, in the healing wounds of the lathyritic animals. PAS-positive material was similar in the wounds of these and normally fed animals, while phosphatase concentration appeared to be slightly higher in the lathyritic rats.

4. The diminished strength in wounds of lathyritic animals may largely be due to a persistent cellularity of the healing tissue, with consequent reduction in the amount of collagen laid down, and partly to an overall thinning of the skin in this condition.

5. The administration of thyroxine to lathyritic rats modified neither the tensile strength nor the histological appearance of healing wounds.

We are indebted to Dr. T. E. Faulkner for statistical advice; and to Mr. W. Muir and Mr. D. Doig for technical assistance.

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SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF ABDOMINO-SCROTAL HYDROCELE

HYDROCÈLE EN BISSAC

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THIS condition was first described by Dupuytren (1834) and called 'Hydrocèle en Bissac'. Lister (1856) recorded the extraordinary clinical appearance of the condition. It is relatively rare and most writers describe only one case. Prather (1942) reviewed the literature and quoted 53 cases including one of his own. Four more have since been described and the case now to be discussed brings the total to 58.

The following is a case history which presents some points of interest:—

CASE REPORT

D. K., aged 17 years, was seen at out-patients with a two-year history of bilateral scrotal swellings and a swelling on the left side of the lower abdomen. Pain was not a feature. There were no urinary symptoms.

PHYSICAL EXAMINATION.—The most striking features were the presence of a large left-sided scrotal swelling and a suprapubic swelling situated slightly to the left of the midline, extending up to the umbilicus and resembling a distended bladder (*Fig.* 744). On palpation it was obvious that the swellings were connected since a fluid thrill could be obtained between them. On the right side there was a small hydrocele.

FIG. 744.—Pre-operative photograph.

AT OPERATION.—The inguinal canal was opened through the usual groin approach by splitting the fibres of the external oblique and dissecting off the attenuated cremaster muscle-fibres and fascial coverings. The hydrocele was now visible extending upwards beneath the arch of the conjoint tendon and downwards into the scrotum (*Fig.* 745). Removal of the sac was facilitated by aspiration of the fluid (approx. 1000 c.c.). The testicle was delivered into the wound. The hydrocele sac stripped easily from the cord and its attachment to the testicle was divided.

FIG. 745.—The inguinal component of the hydrocele exposed at operation.

The abdominal extension proved to be a serous lined cavity extending upwards behind the lower abdominal muscles and in front of the peritoneum. By gentle traction and blunt dissection this part of the sac was removed with ease. There appeared to be no connexion with the peritoneum. Examination of the cord after removal of the hydrocele showed the presence of a small indirect hernial sac, which was dissected off the cord and excised. The defect between the conjoint tendon and inguinal ligament was closed by suturing the conjoint tendon to the inguinal ligament. A small drain was placed in the scrotum and the wound closed.

On the right side a small vaginal hydrocele was excised. There was no hernia present.

Post-operative progress was satisfactory and the patient was discharged on the tenth day.

COMMENT

1. Causation.—The generally accepted theory of the causation of an abdomino-scrotal hydrocele

(Jacobson, 1893; Macewen, 1896; Tanzer, 1935) is that a high infantile hydrocele exists in which the processus vaginalis is obliterated only at the level of the internal ring. Expansion of the proximal end of the hydrocele sac occurs and enlargement takes place between the anterior abdominal musculature

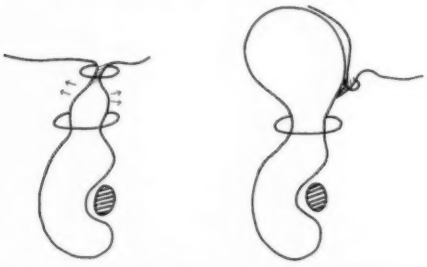

FIG. 746.—The development of an abdomino-scrotal hydrocele according to Jacobson, Macewen, and Tanzer.

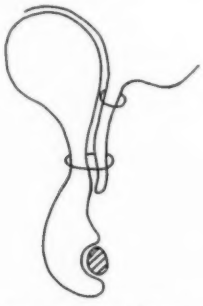

FIG. 748.—Present case, showing the indirect inguinal hernia.

and the peritoneum (*Fig.* 746). Kocher (1878, 1887) and Dupuytren (1834), however, believed that with extreme distension the ordinary hydrocele could push its way though the inguinal canal and assume the abdominal position (*Fig.* 747).

In the present case the inguinal hernia is of interest (*Fig.* 748). Although it is well known that indirect inguinal hernia is often associated with vaginal hydrocele, it is difficult to explain its presence in a case of abdomino-scrotal hydrocele. Three mechanisms of formation seem apparent:—

a. The generally accepted theory of causation of indirect inguinal hernia is that the processus vaginalis remains patent and continuous with the peritoneal cavity through a part, or the whole, of the inguinal canal. In which case, for the formation of a hydrocele the point of occlusion of the processus vaginalis must lie below the level of the internal ring, in or below the level of the inguinal canal. To form the abdominal component, enlargement of the hydrocele must then take place through the inguinal canal, as suggested by Kocher (1878, 1887).

b. That the abdomino-scrotal hydrocele is formed in the manner suggested by Jacobson (1893), and as enlargement takes place a hernial sac is drawn down through the internal ring.

c. That the abdominal component of the sac stretches the internal ring and thus allows a protrusion

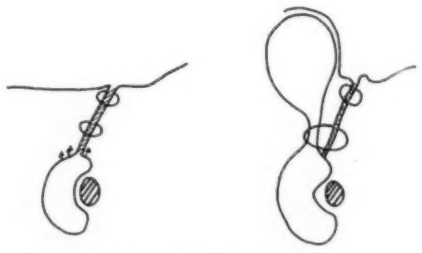

FIG. 747.—The development of an abdomino-scrotal hydrocele according to Kocher and Dupuytren.

of peritoneum to escape down the inguinal canal and form a hernial sac.

Throughout the literature McCune (1948) quotes 7 cases of hydrocele of the canal of Nuck in females with an intra-abdominal extension, and describes an additional case of his own. This particular case revealed the presence of an indirect inguinal hernia, the round ligament lying between the cyst and the hernia.

The practical point arising out of this theoretical discussion is that a hernial sac should be looked for and excised.

2. Excision.—The whole procedure was accomplished through an inguinal incision. This may not always be possible owing to the presence of a thick layer of fascia investing the abdominal component, and an additional lower abdominal incision is necessary (Currie, 1953).

3. The testicle was normal and not atrophied, as occasionally described (Prather, 1942).

I would like to express my thanks to Mr. A. Gourevitch for permitting me to publish this case; and to Mr. A. J. H. Rains for his helpful criticism.

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HÆMANGIOBLASTOMA OF THE BREAST ASSOCIATED WITH PREGNANCY

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MALIGNANT tumours of the breast of vascular origin are rare, 19 cases having been reported in the medical literature. Three cases reported by Patrick, Jarvis, and Miln (1957) were seen over a period of ten years, when 1376 cases of malignant breast tumours were examined. Notwithstanding the frequent occurrence of hæmangiomata in Africans, hæmangioblastoma of the female breast has not previously been described in this racial group. This case is presented both because of the rarity of the condition and the unique initial involvement of both breasts.

CASE REPORT

The patient, an African woman aged 25 years, was admitted to King Edward VIII Hospital, Durban, on July 16, 1957. She had been delivered at home of her second child three months previously and had noticed a lump in her right breast during the last month of pregnancy. The lump was painless and gradually increased in size. Over the ensuing three months further lumps had appeared in this breast, and three weeks prior to admission the patient had felt nodules in the left breast. During this time she successfully breast-fed her newborn baby.

Ten days prior to admission the patient attended a peripheral hospital where the diagnosis of an acute breast abscess was made. The tumour was incised with resulting severe hæmorrhage, for which a blood transfusion of 4 pints was required.

ON ADMISSION.—The patient was pale but well nourished. Her blood-pressure was 116/70 mm. Hg and hæmoglobin 8·4 g. per cent. Both breasts were large, pendulous, and tapering to the nipples. Each breast contained 7–10 nodules, these being both visible and palpable, more or less spherical, and varying in size from 1 cm. to 4 cm. in diameter. The right breast was slightly bigger, and the nipple was retracted. An oblique incision in the upper outer quadrant had broken down and the edges were indurated and inflamed (*Fig.* 749). The floor was composed of friable looking, dark-red material and some blood-clot. To the right and adjacent to the wound was a warm, circular mass based on the wound, measuring 5 cm. in diameter. It was possible to empty the larger masses and see a gradual refilling over the ensuing few seconds. There were a few dilated veins coursing over each breast, but there were no palpable axillary lymph-nodes.

The patient was given a transfusion of 2 pints of blood and on July 19 one of the nodules from the right breast was removed for histological examination. A diagnosis of hæmangioblastoma was made. Initially the patient refused treatment, but after five spontaneous hæmorrhages from the wound of the right breast, necessitating repeated blood transfusions to a total of 12 pints, she agreed to a right simple mastectomy. This was carried out on Aug. 27.

Histological examination of the biopsy and mastectomy specimens showed a neoplasm varying in appearance from an apparently benign angioma to solid areas in which occasional mitoses were seen. In parts the breast acini were replaced by tumour tissue and there was an overall increase in collagen, while in other parts clumps of tumour cells were lying in vascular spaces. The appearances varied from field to field (*Figs.* 750–754).

FIG. 749.—Hæmangioblastoma of both breasts on admission. Incision illustrated necessitated a blood transfusion of 4 pints.

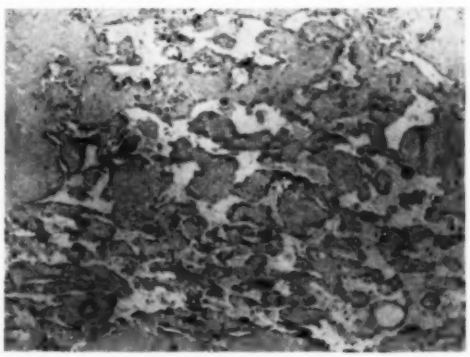

FIG. 750.—Low-power view of an area, showing structure of a cavernous hæmangioma. (× 76.)

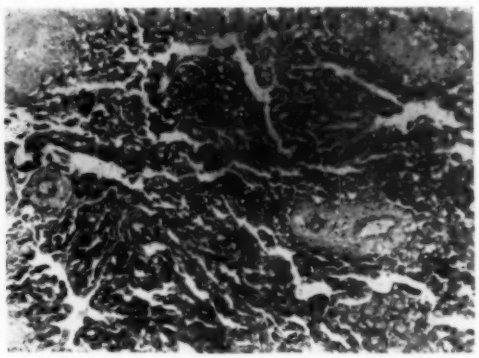

FIG. 751.—Low-power view of an area adjacent to *Fig.* 750, showing a more cellular structure with small vascular channels and suggesting a capillary hæmangioma. (× 76.)

The mastectomy wound healed by first intention, but by Sept. 28 the left breast had increased greatly in size and further subcutaneous nodules had appeared over the right scapula and right deltoid. Both masses were attached to underlying muscle. The liver had enlarged to three fingerbreadths below the costal margin.

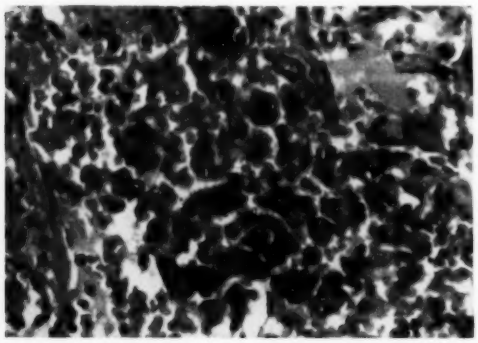

FIG. 752.—High-power view of a cellular area showing clumps of large swollen endothelial cells lying in a vascular sinus. (× 353.)

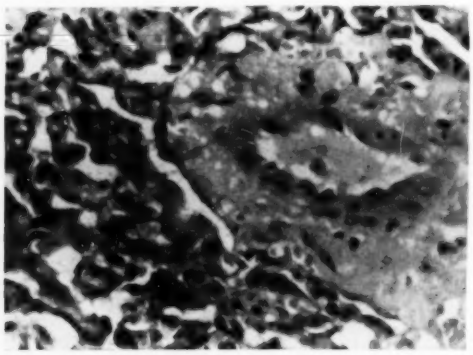

FIG. 754.—High-power view showing a prominent vessel in a cellular capillary hæmangiomatous area. (× 350.)

The patient was anxious to return to her hut and was discharged from hospital on Oct. 21. She died at home three days later. No necropsy examination was carried out.

DISCUSSION

Patrick, Jarvis, and Miln (1957) reported 3 cases, each occurring in young women, and a clear clinical picture of this highly malignant tumour has now emerged. The tumour which had previously been described as 'benign metastasizing hæmangioma' (Robinson and Castleman, 1936), 'angioblastoma' (Enticknap, 1946), 'hæmangiosarcoma' (Mallory, 1949), and 'hæmangio-endothelioma' (Stout, 1943), they designate 'hæmangioblastoma'. The latter term satisfactorily describes a malignant tumour of cells resembling primitive endothelium with a capacity to form vascular spaces.

The tumour occurs in young women, only 2 of the reported cases being in postmenopausal patients. This fact, associated with the oft-times innocent initial clinical features, has in many cases led to a delay in treatment. In addition, the histological features vary considerably from area to area, and a single biopsy may belie the highly malignant nature of the tumour.

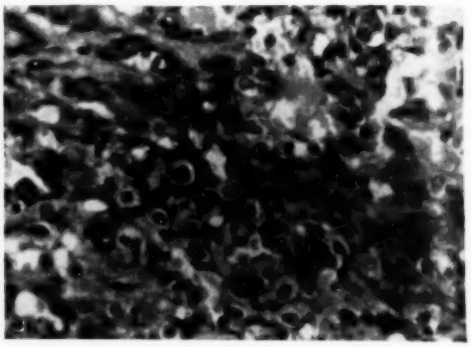

FIG. 753.—High-power view of a cellular area showing large swollen pleomorphic endothelial cells with few vascular channels. (× 350.)

In all the reported cases the disease has run a rapidly fatal course, with widespread dissemination of the tumour. The regional lymph-nodes are not typically involved, even in the advanced stages (Haagensen, 1956). One case survived more than five years and she died of her disease a short while later (Tibbs, 1953). Surgery and irradiation have in all reported cases proved to be of no avail.

In the case presented, the well-documented clinical and histological features were clearly demonstrated, but of particular interest was the initial widespread involvement of both breasts in the absence of clinically detectable secondary deposits elsewhere. Two main possibilities were considered to explain this unusual sequence of events. Either heavy involvement of the highly vascular contralateral breast may have occurred by a tumour which is known to metastasize mainly by the hæmatogenous route, or the tumour was of multifocal origin, occurring independently in both breasts.

This latter possibility would support a view expressed by Shore (1957) that there is a local peculiarity of the female breast in its response to hormonal stimulation predisposing it to malignant changes; a view backed on clinical grounds by the frequent association of the tumour with pregnancy (Tibbs, 1953, Enticknap, 1946), and the high incidence in the reproductive period of life.

SUMMARY

A case of hæmangioblastoma occurring in both breasts simultaneously associated with pregnancy in a young African woman is reported. The clinical picture, course, and rapidly fatal outcome followed the typical pattern of this condition previously described.

I wish to express my thanks to Professor A. E. Kark for advice; to Dr. Wainwright for his pathological report; and to Dr. S. Disler, Medical Superintendent, for permission to publish the report.

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MESONEPHROBLASTOMA OF THE UTERINE CERVIX

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THE following case report concerns the histogenesis of a rare neoplasm thought to have been derived from mesonephric remnants in the uterine cervix, but in some places having a histological appearance indistinguishable from a Wilms' tumour of the kidney. It has been called a 'mesonephroblastoma of the uterine cervix', with the intention of indicating its embryonic nature and its probable origin from residual mesonephric structures.

CASE REPORT

A 53-year-old clerk with no previous history of illness, except for calcific bursitis, sought medical advice for treatment of a serosanguineous vaginal discharge which had been present for three months. When examined she was found to have a soft, lobulated, and pedunculated tumour arising from the endocervical canal. This was readily removed, with the impression that it was either a degenerating fibroid or else a malignant growth. The tumour measured 3 × 2 × 1 cm. and was dark brown in colour and had the consistence of brain tissue. At the same time a complete circumferential cone of the endocervix was removed for histological examination. It is necessary to describe the appearances of the cervix, the pedunculated tumour, and the uterus which was removed at a later operation under separate headings, since these three regions showed the following distinctive and totally different structures:—

Cervix:	Well-differentiated mesonephric adenocarcinoma.
Pedunculated tumour:	Embryonic 'mixed' tumour.
Uterus:	Embryonic carcinoma with glomeruloid structures.

PATHOLOGICAL REPORT.—

Cervix.—The whole of the 360° cervical cone was sectioned and serial blocks were examined. All of these showed the musculofibrous tissue to be widely infiltrated by adenocarcinoma. The tumour was well differentiated and showed no traces of embryonic tissue in any section. It consisted of glandular spaces of varying size separated by normal muscle-bundles. Many of the acini had dilated lumina and many were quite small. The epithelium varied considerably in height, being columnar in a few glands but mostly cuboidal or pseudo-stratified in others (*Fig.* 755). It was distinguished from a carcinoma of endocervical gland origin by its distinctive low cuboidal epithelium and by the failure of the cells to stain with mucicarmine. The appearance was very similar to a drawing from an early article by Meyer (1907), in which he described the first published case of a Gartner's duct carcinoma. It also resembled the structure illustrated in Figs. 7 and 10 in the article by Novak, Woodruff, and Novak (1954) and Huffman's (1948) Fig. 7 of a case of mesonephric carcinoma.

Pedunculated Tumour.—The microscopical appearance of this tumour (*Fig.* 756) bore no resemblance whatever to the mesonephric adenocarcinoma in the endocervical cone. The pedunculated tumour showed the same

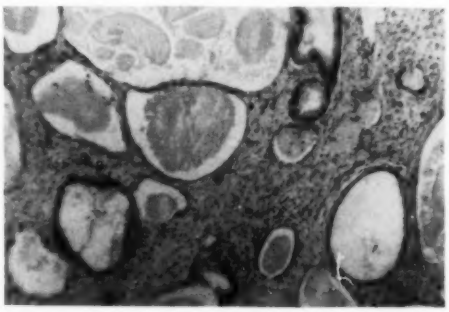

FIG. 755.—Typical section from cervical cone. This is a pure adenocarcinoma with no 'mixed structure'. The epithelium in one or two glands is columnar and the cells have a plentiful amount of clear cytoplasm. In most of the other glands it is cuboidal or stratified. The latter is indistinguishable from that seen so frequently in tubular structures near the hilum of the ovary. (× 80.)

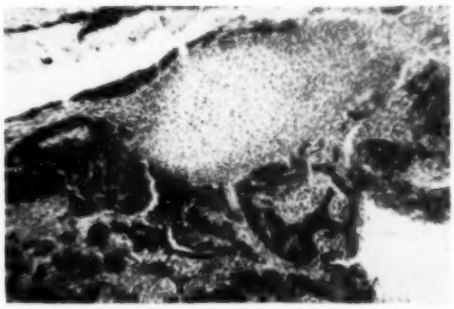

FIG. 756.—Pedunculated tumour. The structure is that of a malignant embryoma with abundant contorted tubules mingled with primitive mesenchyme and is indistinguishable from a Wilms' tumour. (× 15.)

appearance in all areas. It was an embryonic tumour, largely epithelial, with primitive carcinomatous tubules, acini, and solid cellular regions indiscriminately mingled and showing a gradual transition to embryonic sarcomatous mesenchyme. In many places this mesenchyme was producing embryonic cartilage (*Fig.* 757). If its origin were not known, it might easily have been taken for a Wilms' tumour.

Uterus.—Following the pathological examination of the above tissue, a panhysterectomy was performed after irradiation of the cervix. Sections of the lower uterine segment showed that the myometrium was infiltrated right through to the peritoneal coat, in many regions, by an embryonic carcinoma in which there were no sarcomatous elements. Carcinomatous tufts and tubules resembling the pro-glomeruli of a Wilms' tumour were numerous (*Fig.* 758). The broad ligament and the hilum of the ovary on one side contained some very small glandular structures, and it could not be decided whether they were mesonephric rests commonly found in those situations or whether they were carcinomatous metastases.

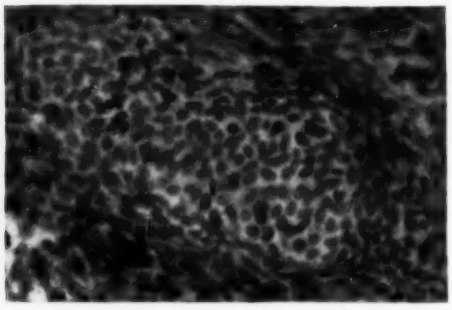

FIG. 757.—Pedunculated tumour. There is a transition from primitive mesenchyme to embryonic cartilage. (× 300.)

FIG. 758.—Myometrium. Embryonic carcinoma. There are several structures resembling pro-glomeruli, each with an invaginated tuft of solid epithelium and a short length of primitive proximal tubule. (× 80.)

COMMENT

In the embryo the mesonephros develops from the intermediate cell mass and forms internal glomeruli as in the metanephros. Its tubules are greatly coiled and open into the mesonephric duct and are at first segmental. Subsequently, secondary nephric units develop between the segmentally arranged ones, so that eventually a large mass of tubules and glomeruli is produced which projects into the abdominal cavity at each side of the mesentery.

The remnants of the mesonephric duct persist in the hilum of the ovary, in the mesosalpinx, and in the cervix, since the duct in its early stages of development courses through the broad ligament close to the uterus and, at the level of the internal os, penetrates the substance of the cervix (Meyer, 1903). Vestiges of the mesonephric duct are found in the adult cervix, and carcinomas of these structures have been described (Huffman, 1948). Such remnants are said to occur in the cervices of 20 per cent of fœtuses and adults, and in some cases these show adenomatous hyperplasia (Wolfe, 1940). It is therefore necessary to distinguish between a simple remnant of this structure and a true neoplasm.

The unusual feature of this case is not the presence of an adenocarcinoma of mesonephric remnants in the cervix, rare enough as this is, but the fact that the tumour had undergone degradation and dedifferentiation to an embryonic state. While it enjoyed the support of the normal cervical stroma, it remained well differentiated. When it began its exophytic growth it apparently found it necessary to produce its own supporting stroma and to do this it reverted to an embryonic state by anaplasia. In this way it became capable of producing embryonic mesenchyme, adenocarcinoma, cartilage, and primitive myxomatous tissue. At the same time it invaded the myometrium and differentiated slightly to produce carcinomatous structures resembling pro-glomeruli.

The features described could only be accounted for by either the persistence of 'embryonic rests' in the cervix for a period of fifty-three years before being stimulated to neoplasia, or else by the development of adenocarcinoma from a mature mesonephric vestige and the subsequent anaplastic degeneration of this growth to a primitive embryonic state. The latter seems the more reasonable and might have taken place in the following steps:—

Cervical mesonephric remnant
Mesonephric adenocarcinoma
Dedifferentiation
Malignant embryonal tumour
Mesonephroblastoma

This process is postulated because of the presence of well-differentiated cervical adenocarcinoma and because of the rarity of an embryonic tumour of Wilms' structure in an adult of fifty-three years.

It has always been accepted that the Wilms' tumour is a derivative of the forerunner of the adult mammalian kidney, the metanephros. It seems not unreasonable to believe, therefore, that a similar tumour could arise from the mesonephric duct since both the mesonephros and metanephros are decended from the same embryonic mesodermal mass.

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INTERSTITIAL-CELL TUMOUR OF TESTIS WITH GYNÆCOMASTIA

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In view of the rarity of these tumours, only some fifty having previously been described in the world literature, it is felt worth while to put on record the following case.

CASE REPORT

W. F., aged 58 years. First seen on Jan. 26, 1957, complaining of pain and swelling in left breast of two months' duration. At first the pain felt as if a hair were being pulled at the nipple. Later, as the swelling appeared, the pain ceased except for one or two tender areas. There had been some soreness in the right breast for a few days.

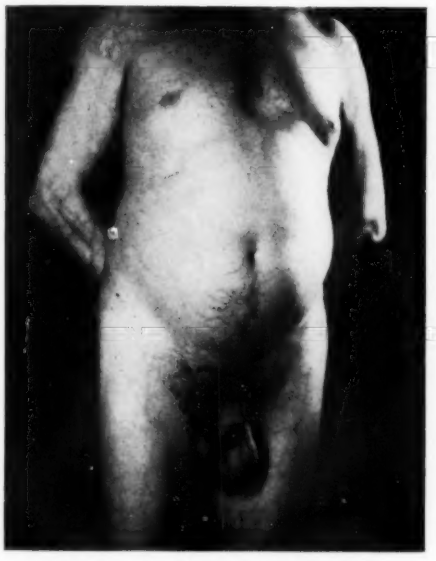

Fig. 759.—Testicular tumour and unilateral gynæcomastia.

Fig. 760.—Three months following operation (note resolution of gynæcomastia).

On Examination.—General condition good. There was obvious increase in size of the left breast, with a firm elongated swelling deep to the nipple. A small mobile left axillary node did not feel significant and there was no abnormality of the right breast. Abdominal examination revealed that the left testis was the site of a tumour measuring approximately 4 × 5 cm. (*Fig.* 759). The tumour was heavy and associated with a small hydrocele, but testicular sensation was still present. The spleen was just palpable, but no other abnormality was made out in the abdomen and further physical examination was negative. Although the tumour had been noted by the patient for about nine years it had been growing very slowly during this time, and the only trauma recalled was a hard kick on the scrotum at the age of 9 or 10 years. Radiography of the chest was negative. W.R., negative. A Hogben test to detect A.P.L. hormone gave a negative result.

Feb. 7: Left orchidectomy performed.

Pathology Report.—"The specimen (*Fig.* 761) consisted of testis, epididymis, cord, and tunica vaginalis. A small effusion was found within the tunica. A solid, ovoid tumour (4 × 5 × 4 cm.) occupied the main part of the body of the testis. This tumour was of greyish-yellow colour and was subdivided into lobules by connective-tissue septa. A thin rim of surviving testicular tissue was present antero-inferiorly. Histologically (*Fig.* 762) the tumour is composed of irregular sheets of large cells with vesicular nuclei and abundant, often faintly eosinophilic, cytoplasm. There is some irregularity of nuclear size and hyperchromasia is occasionally seen. Mitotic activity is present, but it is not a striking feature. There is no attempt at acinar formation. In some foci aggregates of larger cells with small nuclei and abundant cytoplasm of 'foamy' type can be seen—forming a 'hypernephroma-like' pattern. Sections specially stained reveal much fat in many of the tumour cells. Heidenhain's iron-hæmatoxylin shows rod-like structures in some fields and these are thought to be the 'crystalloids' occasionally seen in and around Leydig cells. This tumour is not a seminoma, nor is it a teratoma. It conforms to the pattern of an interstitial-cell tumour of testis, but is apparently rather less well differentiated than the usual interstitial-cell tumour and is therefore to be regarded as an interstitial-cell carcinoma, probably of a rather low grade of malignancy."

Progress.—The patient made an uneventful recovery and was discharged on Feb. 17, ten days after operation. At the time of discharge an obvious decrease in size of the left breast had occurred, although it was still appreciably larger than the right. The patient was seen at intervals for a time following operation and has remained well and free of symptoms since his operation. *Fig.* 760 shows the patient at three months after operation.

FIG. 761.—Cut surface of gross specimen.

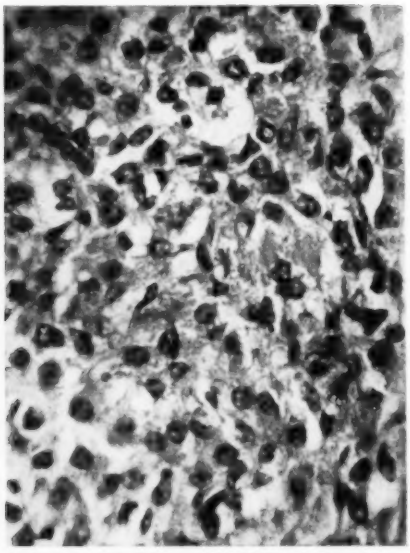

FIG. 762.—Photomicrograph of section of tumour H. and E. (×80.)

DISCUSSION

It is not proposed to review the literature since an excellent review was done by Diamond and Impink (1956). With regard to the changes sometimes found in the secondary sex characters in cases of this type, it is of interest to note that the patient noted no change in libido nor in the rate of growth of beard. He has no children, but this is due to the fact that there was marital trouble with his first wife and by the time of his second marriage he felt he was too old to have children. He did note that the right testis increased in size after removal of the left.

SUMMARY

A case of interstitial-cell tumour of the testis with unilateral gynæcomastia is recorded. No other secondary sex characters were affected. The gynæcomastia disappeared after removal of the tumour.

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AORTOGASTRIC FISTULA

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ŒSOPHAGO-GASTROSTOMY is one of the popular procedures for restoring the continuity of the alimentary tract after resection of the œsophagus. An unusual complication of this operation is the development of a fistula between the stomach and the aorta.

Three fatal cases in which this was encountered are recorded here, and the possible aetiological factors are discussed in conjunction with the previous communications on the subject.

CASE REPORTS

Case 1.—Mrs. E. G., aged 79 years, was admitted to the hospital complaining of indigestion, heartburn, regurgitation for 30 to 40 years and occasional episodes of dysphagia. During the four months before admission her dysphagia had progressed to the point where she was able to swallow only liquids. She had lost considerable weight. Since the onset of dysphagia the heartburn was less distressing.

Radiological barium studies of the œsophagus showed an irregular filling defect in the middle third and a hiatal hernia. Œsophagoscopy showed retention of copious white froth in the œsophagus; a fungating growth almost completely occluding the lumen was seen at 27 cm. The microscopical examination of the biopsy showed the tissue to be gastric mucosa together with masses of œdematous connective tissue infiltrated by adenocarcinoma. The tumour tissue therefore originated from gastric epithelium rather than œsophageal, and this would, in turn, suggest that the tumour had originated in a gastric-lined œsophagus.

On March 28, 1956 partial œsophagectomy and œsophagogastrostomy was performed through a left

thoraco-abdominal approach, with resection of the fifth rib. An extensive growth of the middle third of the œsophagus was found together with a sliding type of hiatal hernia. The stomach was apparently free from growth. The œsophagus was divided at the level of the superior border of the arch of the aorta. The growth was

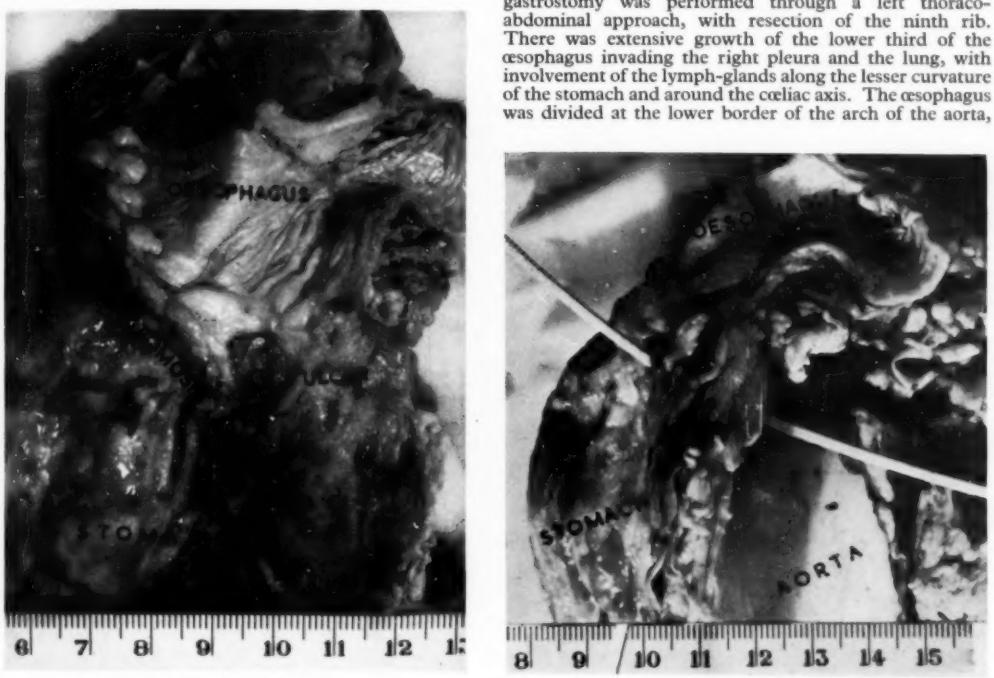

FIG. 763.—Photographs of the post-mortem specimen of *Case* 1. The ulcer is situated across the œsophagogastric anastomosis and a probe is passed through the aortogastric fistula.

excised and continuity restored by œsophagogastrostomy using a separate opening in the stomach. The anastomosis, using chromic catgut, was performed to the left and level with the upper border of the arch of the aorta. The stomach was anchored to the pleura overlying the descending aorta 2 in. below the level of the anastomosis.

The post-operative course was quite uneventful until three and a half weeks later, when she vomited a small quantity of fresh blood. Twelve hours later she had a large hæmatemesis accompanied by shock. A further twelve hours later she had a massive hæmatemesis and immediately died—i.e. twenty-four hours after the onset of her hæmatemesis.

Post-mortem examination showed the stomach to be greatly distended with blood. There was an ulcer 1·2 cm. in diameter situated at the anastomosis opposite the arch of the aorta. In the base of the ulcer was a small fistula communicating with the lumen of the aorta (*Fig.* 763). The aorta itself was healthy except for a few patches of atheroma. At the site of the fistula there appeared to be a small dissecting aneurysm through a plaque of atheroma. The stomach did not show any evidence of neoplasm and the mucosa at the point of the anchoring suture was healthy. Histology of the ulcer showed subacute ulceration with penetration through all muscle coats, with no fibrosis.

Case 2.—Mrs. R. S., aged 70 years, was first seen on Aug. 14, 1956 with a history of progressive dysphagia of four months' duration; she was able to swallow liquids only with difficulty. She had lost a lot of weight during this period. Œsophagoscopy revealed a firm malignant stricture at 34 cm. A biopsy showed a well-differentiated squamous-cell carcinoma.

On Oct. 2 partial œsophagectomy and œsophagogastrostomy was performed through a left thoraco-abdominal approach, with resection of the ninth rib. There was extensive growth of the lower third of the œsophagus invading the right pleura and the lung, with involvement of the lymph-glands along the lesser curvature of the stomach and around the cœliac axis. The œsophagus was divided at the lower border of the arch of the aorta, and the growth was removed along with the upper part of the stomach. Œsophagogastrostomy was carried out through a separate opening in the stomach. The anastomosis was performed to the right and under the arch of the aorta.

The immediate post-operative period was smooth, and the wound healed by primary intention. She was taking a semi-solid diet and was ambulant at the end of two weeks. On the seventeenth post-operative day, at 2 p.m., she collapsed and had a hæmatemesis of 8 oz., accompanied by sudden pain in the chest. Later in the day she passed a large melæna stool and had a further hæmatemesis. Her condition remained critical and the response to resuscitation was poor. Early next morning at 6 a.m. she had a large hæmatemesis and immediately died.

At autopsy the stomach was grossly distended with blood and an ulcer 1·5 cm. in diameter situated across the anastomosis was noted. The base was composed of blood-clot, soft debris, and remnants of catgut suture material used for the anastomosis. At a distance of 2·5 cm. from this ulcer a small gastric erosion was found in the line of the anastomosis, but this did not communicate with the aorta. At the site of the fistula the aorta and the anastomosis were adherent (*Fig.* 764). The aorta showed ulceration and atheromatous changes around the opening of the fistula (*Fig.* 765). Histology at the site of the fistula showed the picture of peptic ulceration. The small area of gastric erosion showed no penetration beyond the submucosa.

Case 3.—Mrs. N. M., aged 49 years, gave a history of progressive dysphagia for three months and loss of 1 st. in weight during this period. Œsophagoscopy was performed and a malignant stricture at 31 cm. from the upper incisor teeth was revealed.

FIG. 764.—Photograph of the post-mortem specimen of *Case* 2. Note the blood-clots and remnants of suture material in the base of the ulcer, which is situated across the anastomosis. There is also an area of gastric erosion with healthy mucous membrane between them.

On July 26, 1957 partial œsophagectomy and œsophagogastrostomy was performed through a left thoracoabdominal approach, with resection of the seventh rib. The growth was found to extend from 2·5 cm. below the arch of the aorta to the level of the diaphragm and was adherent to the opposite pleura. About 1 cm. below the arch of the aorta the œsophagus was divided. The growth was resected together with the upper fifth of the stomach and the spleen. The upper end of the œsophagus was brought to the left of the aortic arch and anastomosed with chronic catgut to the fundus of the stomach at the level of the arch. The anastomosis was found to rest freely on the aortic arch.

The post-operative course was as follows: On the second day she developed some collapse of the left lower lobe and pleural effusion. Half a pint of blood-stained fluid was aspirated from the left pleural cavity on the third day. On the seventh day the left pleural cavity was drained by an intercostal catheter and an anastomotic leak was diagnosed. On the following day copious dark-coloured fluid was aspirated from the markedly distended stomach. On the evening of the twelfth day the patient was very apprehensive and complained of sudden pain in her chest, followed immediately by a massive hæmatemesis and fresh blood discharging through the intercostal catheter. Her condition deteriorated rapidly, and she continued to have repeated hæmatemesis for the next twelve hours, when she died.

Autopsy revealed that the stomach was grossly distended with blood and that there had been a breakdown of the anastomosis. There was an ulcer 2 cm. in diameter across the anastomosis and opposite the aorta. In the base of this ulcer was a fistula 0·5 cm. in diameter communicating with the great vessel. The aorta showed ulceration of the intima around the fistula. Histology of

FIG. 765.—Shows the aortic surface of the fistula and atheromatous plaques in the aorta in *Case* 2.

the ulcer showed complete replacement of the gastric and œsophageal wall by granulation tissue and debris, with very little fibrosis and extension of ulceration to the adventitial coat of the aorta.

DISCUSSION

The development of aortogastric fistula following œsophagectomy and œsophagogastrostomy for carcinoma of the œsophagus has been recorded on two previous occasions.

Merendino and Emerson (1950) reported the first 2 cases. Their first case was a man aged 63 years who had œsophagectomy for carcinoma of the lower third of the œsophagus. The œsophagogastric anastomosis was performed to the right and at the inferior border of the aortic arch. At the time of anastomosis a suture was accidentally placed through the aortic wall. On the twelfth post-operative day the patient experienced a sudden pain in the chest and immediately had a small hæmatemesis. A further hæmatemesis followed, and he died 2 hours later.

Autopsy revealed a fistula between the aorta and the œsophagogastric anastomosis, apparently created by the accidental suture.

Their second case was a man aged 50 years who had a growth at the junction of the lower and the middle third, and the œsophagogastric anastomosis was also placed to the right and underneath the arch

of the aorta. In this case the gastrosplenic ligament was interposed between the aorta and the anastomosis, but not sutured. This was done with the specific purpose of protecting against the possibility of developing aortogastric fistula.

At autopsy the ligament appeared to have undergone dissolution. On the seventeenth post-operative day the anastomosis leaked, followed by empyema. Radiological examinations five weeks after the operation showed stenosis at the site of anastomosis and a blind tract about 1 cm. long extending laterally from it (i.e., towards the aorta). Dilatation was subsequently carried out. Six weeks later the patient was re-admitted with a sudden onset of chest pain associated with hæmatemesis. He died 15 hours later from progressive hæmorrhage. Autopsy showed an ulcer 2 cm. in diameter at the level of the anastomosis on its lateral side communicating with the aorta by an opening that was 6 mm. in diameter.

Brookes and Stafford (1952) reported a case of a woman 59 years of age who had carcinoma of the œsophagus extending from 1 cm. below the arch of the aorta for a distance of 6 cm. Following œsophagectomy the œsophagogastric anastomosis was performed to the left of the aortic arch. The stomach was anchored to the mediastinal pleura over the aorta by a suture. This was well away from the site of anastomosis. On the fourth post-operative day she developed a leak from the anastomosis. The resulting empyema was subsequently drained. On the tenth post-operative day the chest was re-opened and the leak in the anastomosis was repaired. Subsequently there was a further leak and the empyema was showing satisfactory progress. Eight weeks later the patient suddenly collapsed with hæmorrhage through the empyema track and copious hæmatemesis. She died within a few minutes.

Autopsy revealed an aortogastric fistula. At the site where the stomach was anchored to the aorta there was an ulcer; two small erosions in its base had extended directly into the vessel wall.

In considering the aetiological factors direct trauma to the aortic wall is self-evident. It is possible that the tension of anchoring sutures on the stomach may be a factor in causing local ischæmia leading to ulceration which may progress to involve all the coats. However, in *Case* 1 the site of anchoring suture was noted to be healthy.

Immediate relationship and direct contact of the line of anastomosis to the aortic arch seems to predispose to fistula formation. Three were placed to the right and under the arch of the aorta, one at the upper border, and two to the left of it.

Ulceration in the immediate neighbourhood of the anastomosis was a common feature in these cases. Ulceration of the gastric mucosa following œsophagectomy and total vagotomy has been previously observed. In one of the present cases, *Case* 2, there was in addition a superficial erosion.

It would therefore appear that ulceration precedes fistula formation and eventually extends to involve the aorta.

Erosion of blood-vessels is a frequent complication associated with chronic peptic ulceration, and it is likely that the aorta is eroded by the same process, i.e., by the action of hydrochloric acid and digestive enzymes. Ulceration of the jejunum and fistula formation into the aorta has never been described in association with jejuno-œsophagostomy, and this would support the significance of gastric secretion in producing the fistula.

The other contributory factors causing ulceration and imperfect healing of the anastomosis are the impoverished blood-supply at the anastomosis as the result of mobilization of the stomach and œsophagus and infection around the anastomosis—in particular, a localized abscess formation between the suture line and the aorta.

The constant pulsations of the aorta with systolic pressure head may hasten the process of thinning out the ulcer crater. The onset of hæmorrhage in these 6 cases was variable from the twelfth post-operative day to eleven weeks; on an average the interval was 3–4 weeks. If the aortic wall against the ulceration is subjected to trauma, or is in a state of degeneration or involved in the process of inflammation, it is more likely that fistula formation will occur. Atheromatous plaques were noted in *Cases* 1 and 2 at the site of the fistula.

CONCLUSION

The interval between the prodromal hæmatemesis and death from exsanguination was found to vary from a few minutes to 24 hours, with an average of 16 hours. The diagnosis is based upon the recognition of this 'warning' hæmatemesis, accompanied by shock, sudden chest pain, a collapsed state, and a fall in the blood-pressure. Conservative treatment with resuscitation measures has proved unsuccessful in these 6 cases.

It would therefore seem a reasonable suggestion that surgical intervention should be seriously considered. Immediate thoracotomy and repair of the fistula after direct control of the hæmorrhage by partial lateral occlusion of the aorta should be undertaken before the vital organs have suffered irreversible changes from the effect of the blood-loss.

SUMMARY

Three fatal cases due to exsanguination from an aortic fistula following œsophagogastrectomy are illustrated. The literature on the subject is reviewed. The possible aetiological factors are discussed. Direct control of the hæmorrhage by surgical intervention is advocated.

I am very grateful to Mr. J. S. Davidson for his valuable guidance and encouragement; and to Mr. E. R. Lecutier for his criticisms and advice. The cases reported here were under their care and I am indebted for their kind permission to quote them. The photographs were prepared by Mr. P. Harrison.

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SMALL-BOWEL OBSTRUCTION FOLLOWING ANTECOLIC PARTIAL GASTRECTOMY

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It is well recognized that small-bowel obstruction may follow antecolic partial gastrectomy. The cause of obstruction is usually an internal hernia, the small bowel passing through the gap between the anastomosis in front and the transverse colon behind. Stammers (1952, 1954, 1955) first drew attention to this condition by reporting a case of his own and subsequently describing 15 cases that he had collected. He found that these cases fell into five different groups according to the mechanics of the obstruction, and he also described a sixth type of internal hernia associated with the retrocolic anastomosis. On this experience he strongly advocated closure of these artificial gaps with a few sutures.

The case recorded here is a rare form of small-bowel obstruction following the antecolic type of partial gastrectomy, but unassociated with the gap described. The obstruction of the small intestine was caused by a very dilated colon which bulged around the sides of the anastomosis from behind, became adherent to itself in front, and kinked the efferent loop of the anastomosis.

CASE REPORT

The patient, a man aged 80 years, gave a history of obstinate constipation for twenty years following hæmorrhoidectomy. During the previous few months he had developed nausea, flatulence, anorexia, loss of weight, and had vomited on one occasion.

On Examination.—He was found to be a frail, elderly man with abdominal distension and a palpable mass in the upper abdomen. His hæmoglobin was 50 per cent. Barium-meal examination revealed gross irregularity of the distal third of the stomach, mainly on the greater curvature, the appearances being consistent with a neoplasm. A barium enema showed gross gaseous distension throughout the colon and the presence of a redundant loop of sigmoid colon, but no organic obstruction.

At Operation.—A laparotomy was performed on Oct. 29, 1957 through a midline upper abdominal incision. Free fluid was present in the peritoneal cavity, and a large carcinoma of the stomach found to be infiltrating the pancreas, although the liver appeared to be free of metastases. A palliative partial gastrectomy was performed with an antecolic type of gastrojejunal anastomosis.

The post-operative routine of an indwelling Ryle's tube and intravenous therapy was followed for 48 hr. and then discontinued. On the third day, however, he vomited 8 oz. of fluid and his abdomen was distended. A large amount of flatus was passed after an enema had been given. Owing to further vomiting, oral feeding was discontinued and the previous treatment by gastric suction and intravenous infusion recommenced. At no time did he complain of any abdominal pain. On the eleventh post-operative day 40 oz. of fluid were aspirated through the gastric tube and on the thirteenth post-operative day 57 oz. By the eighteenth day gastric aspiration was still excessive and vomiting occurred if it was discontinued. On this account a further laparotomy was performed.

The findings were at first difficult to interpret as the anastomosis appeared to be retrocolic in position. On dissection, however, the transverse colon was found to be adherent to itself in front of the anastomosis (*Fig.* 766 A) and had kinked the efferent limb (*Fig.* 766 B). Apart from that portion immediately behind the anastomosis the colon appeared to be distended with gas throughout its length. After freeing the adherent colon from the anastomosis, an entero-enterostomy between the afferent and efferent loops was performed, and a jejunostomy established. His subsequent progress was uneventful, apart from troublesome constipation requiring frequent enemata. The jejunostomy was only used for a few days and then allowed to close, by which time oral feeding was adequate. Histological examination of the specimen showed it to be a poorly differentiated adenocarcinoma of the stomach.

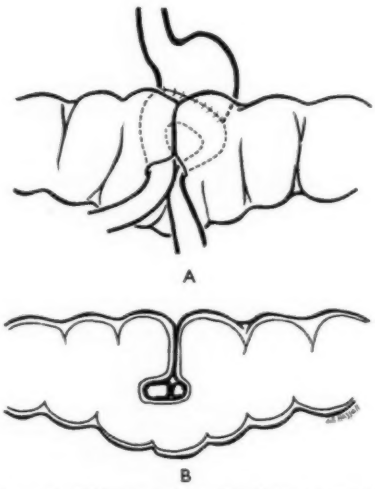

Fig. 766.—Diagram to show distended colon enveloping the gastrojejunal anastomosis (A), and kinking the efferent limb of jejunum (B).

DISCUSSION

The management of post-operative intestinal obstruction is a difficult problem, and Cooke (1958) pointed out the dangers of too conservative an approach and the difficulty in deciding when strangulation of bowel has occurred. The symptoms of post-gastrectomy intestinal obstruction may not be clear cut. Normal gastric emptying following partial gastrectomy may not become re-established for some days, as in the case described by Abel and Cooling (1957) where gastric suction was required for 13 days. In such circumstances it is difficult to decide at which moment to discontinue conservative treatment in favour of operation, and particularly since gastric suction may temporarily relieve any pain. To wait too long may be dangerous. In the case described second laparotomy was delayed until the eighteenth day and then an organic obstruction was found.

SUMMARY

1. Following partial gastrectomy small-bowel obstruction is possible from an internal hernia following the antecolic or retrocolic type of anastomosis.

2. A rare cause of small-bowel obstruction associated with antecolic anastomosis and a dilated atonic colon is described.

I wish to thank Mr. R. W. Raven for permission to publish details of this case under his care. I am also indebted to Miss Hassell, of the Westminster Hospital Photographic Department, for the illustration.

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REVIEWS AND NOTICES OF BOOKS

Chirurgie der Arterien. By Doz. Dr. K. KREMER. 9½ × 6½ in. Pp. 280 + viii, with 150 illustrations. 1959. Stuttgart: Georg Thieme. D.M. 59.

THOUGH arterial surgery requires skilled judgement bred of specialized experience, and involves complicated diagnostic as well as operative techniques, it is still within the scope of the interested general surgeon. A good book devoted to this subject is therefore likely to be in much demand.

This well-produced monograph from Germany is a first-rate contribution. It is difficult to single out any particular chapter for special praise, but the section devoted to coronary insufficiency, the most formidable of remaining challenges to the arterial surgeon, is impressively well done. Throughout, critical evaluation of current thought and methods is combined with a practical approach which is virtually all-embracing: unequivocal advice on management of arterial spasm in connexion with Volkmann's ischæmic contracture, not considered at all, is missed. This small gap apart, the book is highly commended.

Fractures et Luxations. By P. MATHIEU. 8¼ × 5 in. Pp. 211, with 103 illustrations. 1959. Paris: L'Expansion Scientifique Française. 1500 fr.

IT is unusual to find such a complete summary of this subject published in such a small volume.

After a brief general survey, each anatomical area is discussed with particular reference to the most suitable method of treatment and the prognosis.

The text is clear and concise. Line drawings replace radiographs and, as well as illustrating the text adequately, they serve to keep the cost of the book reasonable.

The Pathology and Management of Portal Hypertension. By R. MILNES WALKER, M.S., F.R.C.S., Professor of Surgery, University of Bristol. 9 × 5¾ in. Pp. 113 + viii, with 67 illustrations. 1959. London: Edward Arnold (Publishers) Ltd. 35s.

PROFESSOR MILNES WALKER has written a valuable addition to the monographs that have appeared during the last year or two on portal hypertension. He covers the subject from more aspects than the title would suggest, dealing with the anatomy and physiology, pathology, effects, clinical aspects, management, and treatment of portal hypertension, before describing in considerable detail his methods of doing porta-caval and spleno-renal anastomoses. Direct operations on the varices are then briefly considered. Portal systemic encephalopathy and portal hypertension in children have chapters to themselves and, finally, the results of treatment are discussed.

The value of the book is that it gives Professor Milnes Walker's own personal views and it is not to be expected that contrary opinions and theories will be considered in detail.

In the interests of brevity the number of references is small and those that are given are unfortunately inadequately indexed. For example, Madden's theory on the site of obstruction to the flow of portal blood is taken as an important premise, but no reference is given. This detracts little from the interest aroused by the chapter on pathology. In this the speculative theories to which the author subscribes will undoubtedly form the starting point of much discussion.

The detailed description of the operation of porta-caval anastomosis through the full thoraco-abdominal approach will be of great practical value to all surgeons interested in this complex subject.

Konservative und chirurgische Behandlung angeborener und erworbener Herzfehler. By E. DERRA, Düsseldorf; O. BAYER, Berlin; and H. H. WALTER, Berlin. 8¼ × 5¾ in. Pp. 64 + viii, with 25 illustrations. 1959. Stuttgart: Georg Thieme. D.M. 6.40.

THREE lectures on the management of congenital and acquired cardiac defects, part of a cardiological post-graduate course held in Berlin in May, 1958, have been brought together in this booklet.

The three contributors give a lucid and well-integrated account, covering the entire field in surprisingly few pages. The importance of basic cardiac physiology, especially hæmodynamics, is stressed throughout, even in the excellent chapter devoted to supportive medical therapy. The reviewer foresees a time, probably within this century, when physiology and related sciences will replace remaining empiricism in internal medicine.

Cardiac surgeons will be particularly interested in Professor Derra's advocacy of the direct as opposed to the transventricular approach in pulmonary, and latterly also in aortic, stenosis.

Those who are able to read German will find this little book a valuable and illuminating vantage-point from which to view this subject.

Traité de Technique Chirurgicale. Volume VII. Abdomen. By JEAN QUÉNU, J. LOYGUE, and A. DUBOST. 9¼ × 6½ in. Pp. 770 + viii, with 526 illustrations. 1959. Paris: Masson et Cie. 7200 fr.

THIS is the penultimate of an eight-volume series on surgical technique which now appears in an entirely re-cast second edition, the first having been produced in

1944. This volume deals with biliary, pancreatic, and splenic operations, the abdominal vessels and nerves, abdominal trauma, intestinal obstruction, and peritonitis.

The text is completely up to date, as illustrated by detailed accounts of the most modern techniques of right hepatic lobectomy, pancreatectomy, porta-caval anastomosis, resection and graft replacement of the aorta, and endarterectomy.

As one would expect in a surgical text-book from the country of Mallet-Guy, the section on gall-bladder surgery is particularly good and includes full details of operative cholangiography, although here illustrative radiographs might have been usefully included. Here and there one comes across procedures which appear strange to a reader in this country, for example hepatopexy, splenopexy, and implantation of the proximal stump of pancreas into a jejunal loop after resection of the tail.

References are given at the end of each chapter but British authors are rarely mentioned; an account of abdominal injuries without reference to Gordon-Taylor seems naked!

These are, however, minor criticisms of a well-written, beautifully produced, and profusely illustrated book.

Clinical Neurosurgery, The Proceedings of the Congress of Neurological Surgeons, Washington, D.C., 1957. Volume 5. 9×5¾ in. Pp. 206+xx, illustrated. 1958. Baltimore: Williams & Wilkins Co. (London: Baillière, Tindall & Cox Ltd.). 64s.

THIS is the fifth volume of the proceedings of the congress of neurological surgeons held in the United States and records the meeting in Washington, D.C., in November, 1957. It is dedicated to Francis Grant, of Philadelphia, and the majority of the collected papers of which it is composed deal with neurological surgery in children. A brief biographical sketch of Dr. Grant by G. M. Austin and a bibliography of his published papers is followed by two papers of which he is the part author. "A Clinical Study of Two Hundred Gliomas in the Posterior Fossa of Children" is written in conjunction with R. K. Jones; "Experiences with Cordotomy" with Francis A. Wood; while Dr. Grant is the sole author of "The Surgery of Meningiomas at the Base of the Brain". The remainder of the book is composed of a further eight chapters by several contributors who write on various neurological and neurosurgical problems, of a list of the names and addresses of members who attended the congress, and of an index. Among much that is of interest, the limitations and complications of cordotomy, both low and high, unilateral and bilateral, are noteworthy; motor complications of some significant degree occurring in roughly 20 per cent of patients who have had bilateral operations, and bladder paralysis, usually of lasting character, being the rule in these also. The book is of similar appearance and of much the same size as its predecessors and neurologists and surgeons who practise in this field will want to have it in their bookshelves to complete a remarkable series.

Nouvelle Pratique Chirurgicale Illustrée. Edited by JEAN QUÉNU. 10½×7 in. Pp. 296, with 254 illustrations. 1958. Paris: Gaston Doin et Cie. 3500 fr.

THIS is the twelfth volume of a series published under the editorship of Professor Jean Quénu and providing a pictorial atlas of a variety of surgical operations which are apparently selected and arranged at random.

The present volume deals with the following: (1) Middle-lobe lobectomy for tuberculoma (Claude Dubost); (2) End-to-side porta-caval anastomosis for portal hypertension (Claude Dubost); (3) Excision of a large retroperitoneal pararenal lipoma (Louis Quénu); (4) Prevention of bone and joint tuberculosis by excision of infected lymphatics (R. Kaufman); (5) Modified Manchester operation for repair of uterovaginal prolapse (M. Chartier); (6) Pollicization of the ring finger by Lelac's method (J. Gouzon); (7) Surgical treatment of hallux valgus by excision of the base of the first phalanx (J. P. Dejouany).

Each chapter begins with one or two pages of letterpress describing briefly the indications and scope of the operation. Then follows a series of excellent full-page black-and-white line drawings illustrating the successive steps of it with great detail and clarity. Some indeed may think that there is too much detail and that there is not much value in a full-page drawing of tetra towels being applied, or of a disembodied hand touching an artery forceps with a diathermy needle! Nevertheless the presentation as a whole gives a very clear idea of the methods described (on the whole fairly standard ones), but its value is surely diminished by the haphazard arrangement and choice of operations.

The book is paper-backed and the paper is of poor quality by British standards. There are a few typographical errors.

Transplantable and Transmissible Tumors of Animals. By HAROLD L. STEWART, M.D., KATHARINE C. SNELL, LUCIA J. DUNHAM, M.D., SAMUEL M. SCHYLEN, M.D. Laboratory of Pathology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland. Section XII—Fascicle 40 of the Atlas of Tumor Pathology. 10½ × 7½ in. Pp. 378, with 287 illustrations. 1959. Washington, D.C.: Armed Forces Institute of Pathology. $3.50.

THIS is Section XII—Fascicle 40 of the *Atlas of Tumor Pathology* published by the American Armed Forces Institute of Pathology. The whole work is a superb atlas of pathology beautifully illustrated and documented.

The present volume is in line with the former ones and contains a wealth of material that would be impossible to find elsewhere. The illustrations, and especially the photomicrographs, are magnificent, especially the coloured ones.

The book will appeal not only to pathologists but also to all those who are interested in cancer research. As a reference volume it could not be bettered.

Physiology of Spinal Anesthesia. By NICHOLAS M. GREENE, B.S., M.A., M.D., Professor of Anesthesiology and Lecturer in Pharmacology, Yale University School of Medicine. 9×5¾ in. Pp. 195+xii, with 7 illustrations. 1958. Baltimore: The Williams & Wilkins Co. (London: Baillière, Tindall & Cox Ltd.). 48s.

THIS book is something more than an account of the physiological disturbances consequent on spinal anæsthesia. It is a comprehensive guide to those aspects of physiology with which the anæsthetist who uses spinal anæsthesia should be conversant.

It contains chapters on the nervous system, the cardiovascular system, pulmonary hæmodynamics, hepatic, renal, and endocrine function, the digestive system, metabolism, and obstetric physiology.

The section on cardiovascular physiology is crystal clear. No one who sets out to produce controlled hypotension with the aid of spinal anæsthesia can fail to profit by its analysis of the many factors involved. The chapter on obstetric physiology is clearly the fruit of wide experience. It makes plain many things which have puzzled the young anæsthetist.

The other sections deal in a well-balanced way with such matters as respiratory failure occurring during anæsthesia, a comparison of the effects of spinal and general anæsthesia

on the function of the liver, the effects of sympathetic denervation on renal blood-flow, the relationship of spinal anæsthesia to 'shock' and its place in the diagnosis and treatment of spastic intestinal obstruction. The author adopts a properly sceptical attitude to the advantages attributed to spinal anæsthesia in the diagnosis and treatment of vasospastic conditions; and while he lays the foundations for an understanding of some of the neurological sequelæ of subarachnoid block he does not deal with their pathology.

The author has read widely, and his review of the literature is comprehensive but discriminating. He has the gift of writing pleasantly, in precise and unambiguous English which is nevertheless entirely unpedantic. The book is a welcome contribution to the literature of anæsthesia.

Manuel de Traumatologie. By GEORGES RIEUNAU. 10½×7 in. Pp. 220, with 107 illustrations. 1959. Paris: Masson et Cie. 3000 fr.

THIS book is based on the personal experience of the author over a period of twenty-five years and has been written for the guidance of undergraduate and postgraduate students. The title is misleading, for the book does not mention such fundamental aspects of traumatic surgery as resuscitation, estimation of blood-loss, adequate transfusion, or the coexistence of multiple injuries. Instead it deals purely and simply with the treatment of fractures of the limbs, spine, shoulder, and pelvis. The problem of skin loss in compound fractures is virtually ignored and there is no mention of the torsion-avulsion type of injury or its management.

The methods of treatment advocated are very clearly explained in the text, which has a brief didactic style not unlike that of Professor Lee McGregor's classic on Surgical Anatomy. The accompanying diagrams and drawings are a model of clarity and simplicity.

The author has an obvious personal interest in internal derangements of the knee-joint and a whole chapter is devoted to lesions of the meniscus. The author is a strong advocate of contrast arthrography and has elsewhere reported a personal series of more than 800 cases investigated in this way. By contrast it is rather surprising to find that only 9 pages are devoted to injuries of the ankle and that no mention is made of Bonnin's study of these lesions. Similarly the section devoted to injuries of the hand is scrappy and very disappointing.

There are many references in the text to well-known British and American authors. Many French authors are also named, some of whom are not well known in this country but whose work would appear to be well worth further study. It is therefore a pity to find that there is no bibliography.

This book cannot be regarded as a comprehensive outline of the treatment of fractures, but within its own very carefully defined limitations it gives an accurate description of techniques which would be generally accepted in this country.

Surgery of the Colon. By E. S. R. HUGHES, M.D., M.S.(Melb.), F.R.C.S.(Eng.), F.R.A.C.S., Hon. Surgeon, The Royal Melbourne Hospital, Melbourne. 9¾×6¾ in. Pp. 416+viii, with 234 illustrations. 1959. Edinburgh: E. & S. Livingstone Ltd. 63s.

THERE are many books devoted to the surgery of the colon, but there is always room for others, especially if they add to one's knowledge of this important branch of surgery. Mr. Hughes has already published a monograph on the surgery of the anus, anal canal, and rectum, and this present monograph may be looked upon as a sequel to the former one.

The outstanding feature of this book is the fact that it represents the author's own method of treating colonic diseases. The city of Melbourne has always had a reputation for surgeons interested in the colon ever since the published works of Sir Hugh Devine, and it is pleasing to note that the author has included a photograph of that great surgeon as a frontispiece to his book.

This volume is comprehensive and includes chapters on chronic constipation and chronic diarrhœa as well as those on amœbiasis and actinomycosis of the colon. There is an excellent chapter on injuries to the large bowel which includes gunshot wounds of the abdomen involving the colon. The various techniques which are used in these traumatic cases are well described and illustrated.

The problem of ulcerative colitis is well presented and the various forms of operative procedure are well portrayed.

As would be expected, a large part of this monograph is devoted to the diagnosis and treatment of cancer of the colon. The author's extensive experience in this branch of surgery can be seen in the able way he deals with his cases.

We would like to repeat what we said when we reviewed Mr. Hughes's former monograph: "We would like to congratulate the author on the excellence and clarity of the illustrations in this book, especially the line drawings, the majority of which are original."

Shakespeare and Medicine. By R. R. SIMPSON, M.B., Ch.B., F.R.C.S. 8½ × 5½ in. Pp. 267 + viii, with four plates. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 25s.

THIS is a book which will make a strong appeal to the medical profession for there are many doctors who are very fond of all that Shakespeare stands for.

Shakespeare used doctors as characters in several of his plays and he certainly was conversant with the common practices of medicine as appertained in his day and generation.

Mr. R. R. Simpson has written a very useful reference book on Shakespeare and Medicine and we feel sure it will prove very popular.

The price of this book is most moderate.

Orthopædic Surgery. By Sir WALTER MERCER, M.Ch. Orth., F.R.C.S., F.R.S. (Edin.), Professor Emeritus, Orthopædic Surgery, University of Edinburgh. Fifth edition. 9 × 5¾ in. Pp. 1075 + xii, with 422 illustrations. 1959. London: Edward Arnold (Publishers) Ltd. 90s.

THE new fifth edition of this well-known work has been very thoroughly revised and in places entirely rewritten. Many new illustrations have been added.

There can be little doubt that this edition will prove as popular as the former editions, for it sets out in an interesting and practical way the large and extensive field of orthopædic surgery. Sir Walter is a versatile surgeon who can turn his hand to any specialized branch of surgery and has done so on many occasions.

We congratulate Sir Walter on an excellent production.

The Hand: Its Anatomy and Diseases. By JOHN J. BYRNE, M.D., Professor of Surgery, Boston University School of Medicine. 9 × 6 in. Pp. 384 + xiv, with 163 illustrations. 1959. Oxford: Blackwell Scientific Publications. 80s.

MANY books in the past have been written about the hand but there is always room for others if they contribute something fresh to our knowledge.

This monograph covers all phases of diseases of the hand. The anatomy and development of the hand are presented in a comprehensive and detailed manner but congenital anomalies are not considered. The non-inclusion of congenital anomalies is perhaps a good thing, as A. J. Barsky recently presented the medical profession with an excellent monograph on this subject.

The infections of the hand, including the palmar spaces, are considered in detail. The many and varied forms of trauma and other injuries of the hand are dealt with in an interesting and practical way.

To-day resident medical officers, especially those detailed for casualty duty, will find this book most useful.

Treatment of Cancer and Allied Diseases. Tumors of the Nervous System. Edited by G. T. PACK, M.D., F.A.C.S., Attending Surgeon, Memorial Centre for Cancer and Allied Diseases, New York; and IRVING M. ARIEL, M.D., F.A.C.S., Associate Clinical Professor of Surgery, New York Medical College. Second edition. 10 × 6¾ in. Pp. 316 + xviii, with 340 illustrations. 1959. London: Pitman Medical Publishing Co. Ltd. £5 5s.

THIS is a clearly written monograph which is beautifully illustrated with a large number of original drawings; many of them depict actual operative procedures.

The opening pages of the book are devoted to a general discussion of the major approaches to treatment before considering specific neoplasms.

The volume contains a vast amount of information on the clinical management of patients with every known type of cancer of the nervous system.

The matured clinical acumen of the editors has resulted in a clear and concise volume in spite of the fact that some thirty authorities in the ever expanding field of neuro-surgery and radiotherapy have contributed to the book.

As a book of reference dealing with cancer of the nervous system it could not be bettered.

Breast Cancer. Factors Modifying Prognosis. By A. J. DELARIO, M.D. 9 × 5¾ in. Pp. 208 + xvi. 1959. New York: The Macmillan Co. 52s. 6d.

THIS is an interesting monograph because it presents a factual study of various treatments used on 465 patients admitted for breast cancer to a New Jersey hospital from 1934 to 1951 inclusive.

Dr. Delario's data represent a 100 per cent follow-up of all 465 patients. The best form of treatment in early cases of breast cancer was found to be radical excision without any form of radiation.

There are some 137 tables giving the various forms of treatment and the survival rates without recurrences.

Anyone interested in breast cancer should have a copy of this book for reference.

Textbook of Surgery. Edited by H. FRED MOSELEY, M.A., D.M., M.Ch. (Oxon.), F.A.C.S., F.R.C.S. (Eng.), F.R.C.S.(C.), Assistant Professor of Surgery, McGill University. Third edition. 10 × 6¾ in. Pp. 1336 + xxiv, with 738 text illustrations and 108 colour plates. 1959. St. Louis: C. V. Mosby Co. (London: Henry Kimpton). £6 7s. 6d.

THE third edition of this well-known text-book of surgery has been completely revised and in parts rewritten. It portrays the present-day surgery as practised and taught in Canada and the United States. There is little difference as far as we can see between the technique of surgery as pursued on either side of the Atlantic. Throughout the book the illustrations are good, especially the coloured plates. The author seems to have the happy knack of obtaining the services, as contributors, of the best surgeons in Canada and the United States.

Surgical Pathology. By LAUREN V. ACKERMAN, M.D., Professor of Surgical Pathology and Pathology, Washington University School of Medicine, St. Louis; and HARVEY R. BUTCHER, jun., M.D. Second edition. 9¾ × 6½ in. Pp. 1096, with 1114 illustrations. 1959. St. Louis: C. V. Mosby Co. (London: Henry Kimpton). £5 12s. 6d.

IT is only six short years since the first edition of this work appeared and in the interval the authors have managed to prune and revise the whole text. For a text-book of surgical pathology the best combination of authorship is that of a pathologist and a surgeon working together; and that is what we have in this book.

The outstanding feature of this work is the large number of illustrations, some 1114 in all. The illustrations are excellent and could not be bettered, especially the photomicrographs.

There can be little doubt that the second edition of this work will still continue to be the standard book of reference on surgical pathology.

Fracture Surgery. A Textbook of Common Fractures. By HENRY MILCH, M.D., Consulting Orthopædic Surgeon, Hospital for Joint Diseases, New York; and ROBERT AUSTIN MILCH, M.D., Assistant Resident Surgeon, Peter Bent Brigham Hospital, Boston. 10 × 6½ in. Pp. 470 + x, with 671 illustrations. 1959. New York: Hœber-Harper (London: Pitman Medical Publishing). £7.

THIS book covers the whole field of fracture problems. In this traumatic age it is essential that the diagnosis and treatment of fractures should be practical and competent.

The book itself is divided into four sections: The first is devoted to general considerations, in which the value of plaster-of-Paris is placed in a proper light.

Dr. Herbert Dubovsky has written an interesting chapter on regional anæsthesia in fracture treatment. The second section deals with the axial skeleton; fractures of the vertebral column and of the thoracic cage are treated in a very practical way.

In section III fractures of the upper extremity are considered in detail and many excellent radiographs are included.

The fourth section deals with fractures of the lower extremity.

The book is carefully planned for quick and ready reference; it should prove of real value to residents who have to treat emergency fractures.

Surgical Technique. By STEPHEN POWER, M.S., F.R.C.S., Senior Surgeon to the Dreadnought Hospital, Greenwich. Second edition. 7¼ × 4¾ in. Pp. 411 + x, with 198 illustrations. 1959. London: William Heinemann. 40s.

THIS small monograph is well suited for the house surgeon and the resident surgical officer, for it supplies a wealth of practical surgical technique which is in use in our hospitals.

To-day when medical students have a very comprehensive curriculum to cover many of them on qualification feel the need of a small pocket book on surgery and surgical proficiency and technique.

The second edition of this book has been thoroughly revised and considerable additions have been incorporated. The illustrations, numbering nearly 200, are, with very

few exceptions, original and will prove valuable to the reader.

We are convinced that this second edition will prove even more popular than the first.

Intracardiac Phenomena. By ALDO A. LUISADA, M.D., Director, Division of Cardiology and Associate Professor of Medicine, The Chicago Medical School; and CHI KONG LIU, M.D., Chief, Laboratory of Catheterization and Associate of Clinical Medicine, The Chicago Medical School. Second edition. 10 × 6¾ in. Pp. 179+x, with 82 illustrations. 1958. New York and London: Grune and Stratton, Inc. $9.50.

PART of the material presented in this monograph was published in 1958 under the title *Cardiac Pressures and Pulses*. The authors state that a revision was imposed by the development of left heart catheterization. The techniques of this latter included are those of transbronchial puncture, of puncture through the posterior chest wall, and of suprasternal puncture. Unfortunately percutaneous direct puncture of the left ventricle (first published two years before this second edition) is not mentioned at all; neither is percutaneous catheterization of the left ventricle and aorta, and hence nothing is said of the information to be obtained in this way from pressure withdrawal records. This means that little information is given about the modern studies of aortic stenosis.

Apart from this omission the book is comprehensive and gives full accounts of all technical details of cardiac catheterization, with descriptions of the pulse and pressure records both normal and abnormal. It also describes intracardiac electrocardiograms and phonocardiograms. Presentation of the technical details of analysis of blood gases and of formulæ used in cardiac catheterization make for completeness that virtually makes the volume a manual of cardiac catheterization, even though the authors state this is not their aim.

Bilateral Polycystic Disease of the Kidneys. By O. Z. DALGAARD. Copenhagen: Einar Munksgaard. Kr.29

IN the study of a disease which is admittedly incurable the investigator must have a high sense of the academic importance of his subject, and must be prepared to enter into a minute discussion of the genetic factors concerned as well as the clinical features. In this comprehensive study, which includes a follow-up of 284 patients and their families, there is ample evidence of intensive research. Regarding treatment the author appears to be unconvinced of the value of Rovsing's operation, but rightly takes the attitude of the scientist who demands controls rather than the enthusiastic surgeon. He has some illuminating remarks on the practical implications of eugenic measures.

This book is worthy of close study by all who are interested in the condition.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

Hernia. By Sir HENEAGE OGILVIE, K.B.E., M.A., M.Ch., M.D., F.R.C.S., Consulting Surgeon, Guy's Hospital. 8½ × 5½ in. Pp. 135, with 51 illustrations. 1959. London: Edward Arnold (Publishers) Ltd. 28s.

Principles and Practice in the Management of Surgical Patients. By A. VENUGOPAL MUDALIAR, M.S., F.A.C.S., F.I.C.S., Honorary Surgeon, General Hospital, and Honorary Professor of Clinical Surgery, Madras Medical College. 8½ × 5½ in. Pp. 500 + xii, with 82 illustrations. 1959. Madras: G. S. Press. Rs.20.

A System of Orthopaedics and Fractures. By A. GRAHAM APLEY, M.B., B.S., F.R.C.S., Consultant Orthopædic Surgeon, Rowley Bristow Orthopædic Hospital, Pyrford, Surrey. 8½ × 6½ in. Pp. 357 + viii, fully indexed. 1959. London: Butterworth & Co. (Publishers) Ltd.

Smoking and Lung Cancer. By T. W. LEES, M.D., Law Hospital, Carluke, Lanarkshire. 8½ × 5½ in. Pp. 32. 1959. Published by the Author.

British Medical Bulletin. Volume 15, Number 2, May 1959. Blood Groups. 11 × 8½ in. Pp. 174 + xii. Published by the Medical Department, The British Council. 20s.

Plaies de la Main. By R. SOUQUET and A. R. CHANCHOLLE, The Faculty of Medicine, Toulouse. 9½ × 6 in. Pp. 293, with 131 illustrations. 1959. Paris: G. Doin & Cie. 3600 fr.

Autolyse-Krankheiten in der Chirurgie. By L. KOSLOWSKI. 9½ × 6½ in. Pp. 159 + viii, with 60 illustrations. 1959. Stuttgart: Georg Thieme. D.M. 19.80.

Surgery of the Prostate. By HENRY M. WEYRAUCH, M.D., F.A.C.S., Clinical Professor of Surgery, Stanford University School of Medicine. 10 × 6½ in. Pp. 535 + xii, with 288 illustrations. 1959. Philadelphia and London: W. B. Saunders Co. Ltd. 105s.

Surgery of the Stomach and Duodenum. By CLAUDE E. WELCH, M.D., D.Sc. (Hon.), Visiting Surgeon, Massachusetts General Hospital. Third edition. 8½ × 5½ in. Pp. 405 with 83 plates. 1959. Chicago: Year Book Publishers Inc. (London: Interscience Publishers, Ltd.). 74s.

The Mast Cells. By JAMES F. RILEY, M.B., Ch.B., M.D., Ph.D., D.M.R.T., F.R.C.S.E., Consultant Radiotherapist, Royal Infirmary, Dundee. 9½ × 6½ in. Pp. 182 + x, with 65 illustrations. 1959. Edinburgh & London: E. & S. Livingstone Ltd. 30s.

The Effect of Advancing Age upon the Human Spinal Cord. By L. RAYMOND MORRISON, M.D., with the collaboration of STANLEY COBB and WALTER BAUER. 11 × 8¼ in. Pp. 127 + x, fully illustrated. Cambridge, Mass.: Harvard University Press (London: Oxford University Press). 45s.

Quadri Arteriografici. By E. MALAN and collaborators. 11¼ × 8 in. Pp. 124, fully illustrated. 1959. Torino: Edizioni Minerva Medica. L.5000.

An Introduction to Surgery for Dental Students. By R. P. JEPSON, F.R.C.S., Professor of Surgery, University of Adelaide; and B. N. CATCHPOLE, F.R.C.S., Lecturer in Surgery, University of Sheffield. 8½ × 5½ in. Pp. 166 + x, with 53 illustrations. 1959. London: The English Universities Press Ltd. 21s.

A Short Practice of Surgery. Eleventh edition. By HAMILTON BAILEY, F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., F.R.S.E., MCNEILL LOVE, M.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S., F.I.C.S. With chapters by JOHN CHARNLEY, F.R.C.S. (Eng.), WILLIAM CLELAND, M.R.C.P. (Lond.), F.R.C.S. (Eng.), and GEOFFREY KNIGHT, F.R.C.S. (Eng.). 9¾ × 6¾ in. Pp. 1389 + xii, with 1697 illustrations, 285 in colour. 1959. London: H. K. Lewis & Co. Ltd. £4 4s.

Early Diagnosis. Edited by HENRY MILLER, M.D., F.R.C.P., Physician in Neurology, Royal Victoria Infirmary, Newcastle upon Tyne. 8⅝ × 5⅝ in. Pp. 400 + viii. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 25s.

Lokalanästhesie und Lokalanästhetika. Edited by Prof. Dr. HANS KILLIAN. 10¼ × 6¾ in. Pp. 772 + xx, with 341 illustrations. 1959. Stuttgart: Georg Thieme. D.M. 154.

Die Behandlung van Hiatusheinien und Refluxosophagitis mit Gastropexie und Fundoplicatio. By R. NISSEN and M. ROSSETTI. 9½ × 6½ in. Pp. 153, with 56 illustrations. 1959. Stuttgart: Georg Thieme. D.M. 49.50.

Sympathectomy—An Anatomical and Physiological Study with Clinical Applications. By P. A. G. MONRO, M.A., M.D. (Cantab.), M.Sc., Lecturer in Anatomy, The University of Cambridge. 9¼ × 6 in. Pp. 290 + xx, with 84 illustrations. 1959. London: Oxford University Press. 75s.

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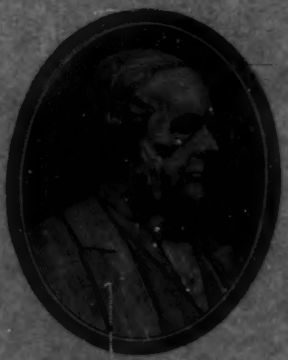

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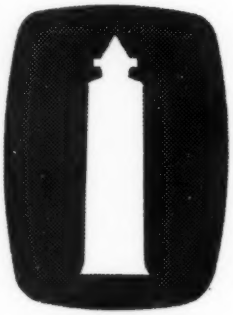

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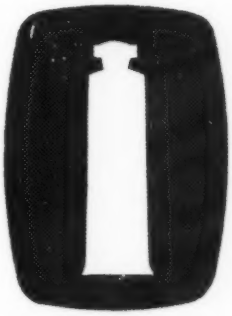

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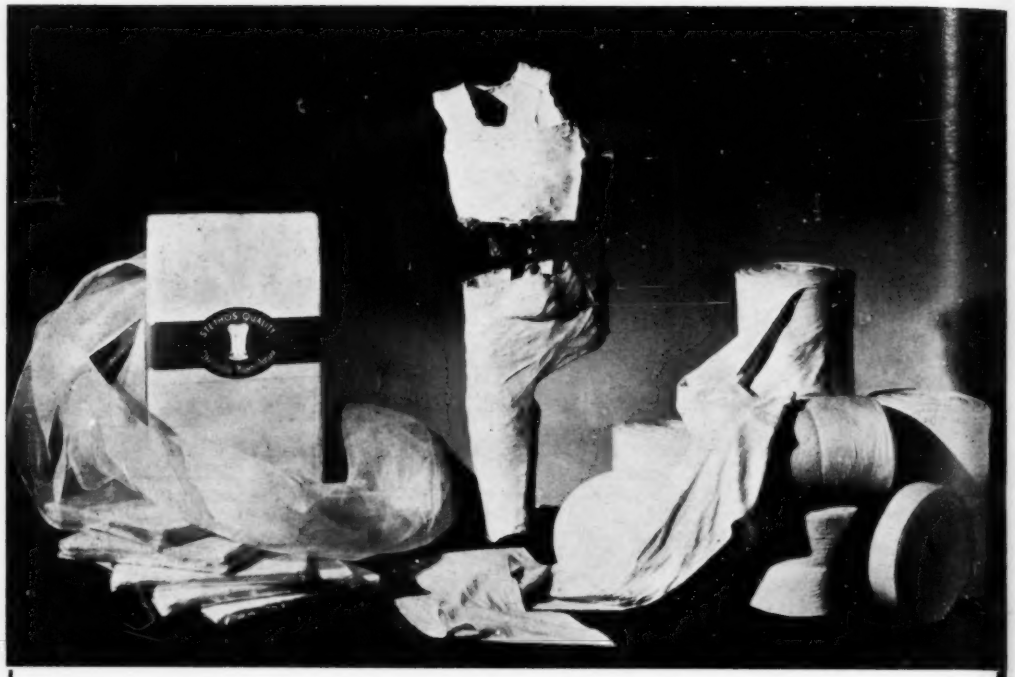

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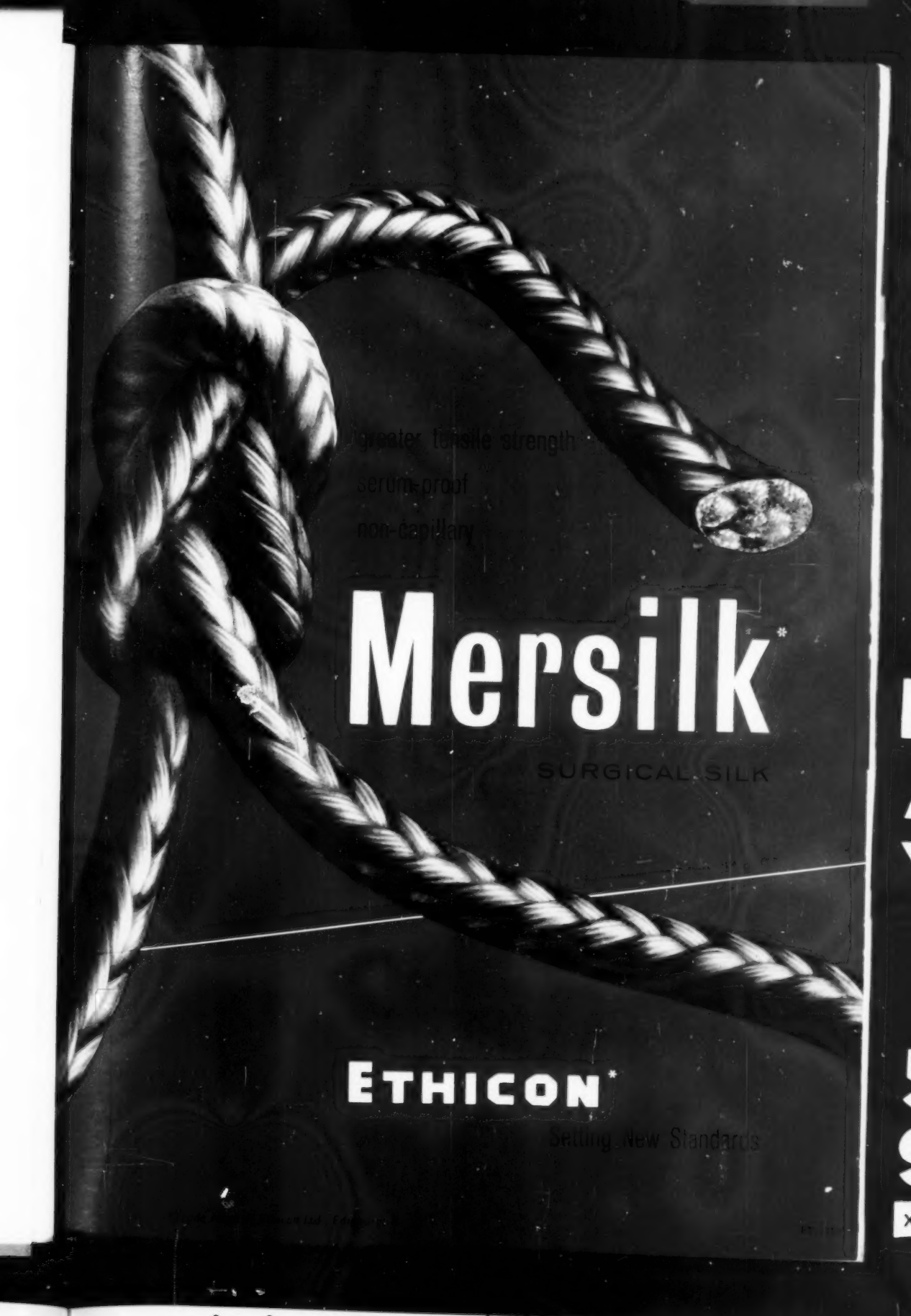
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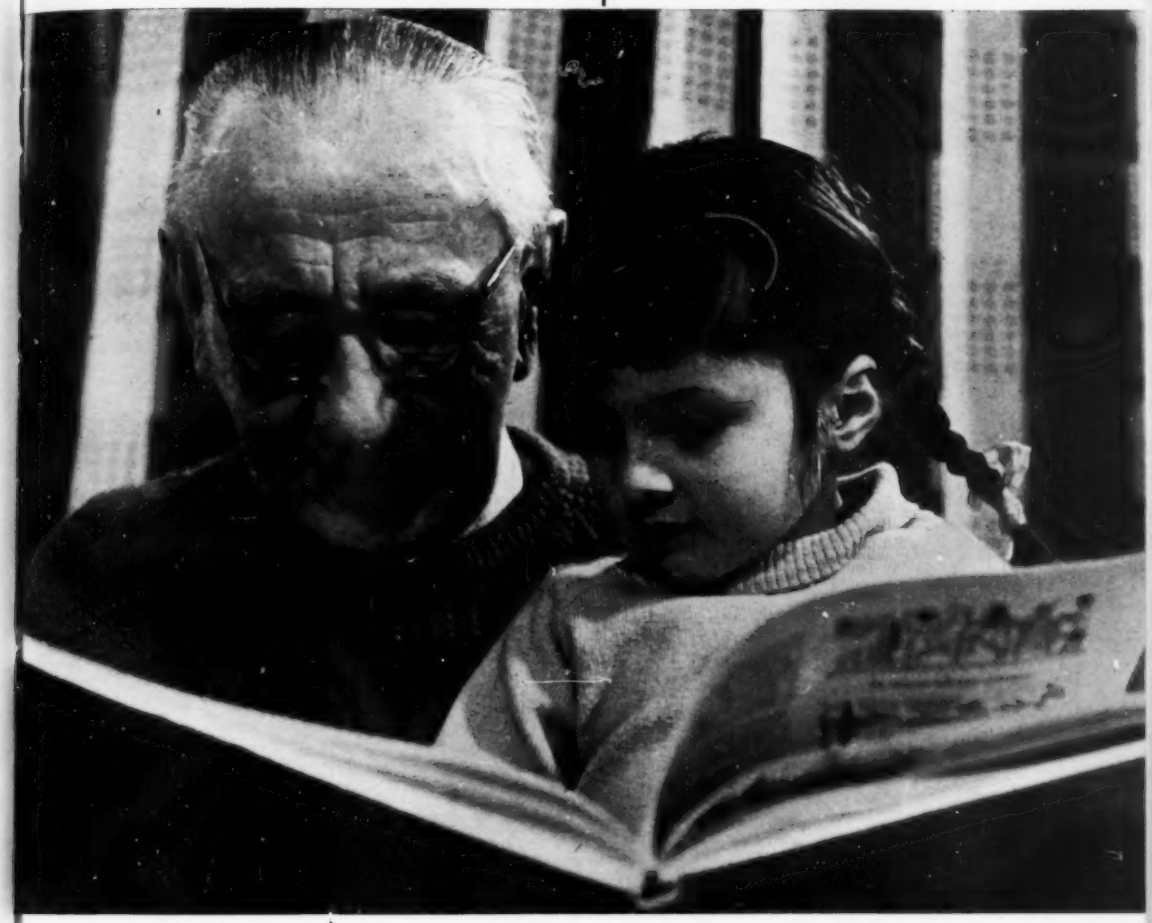

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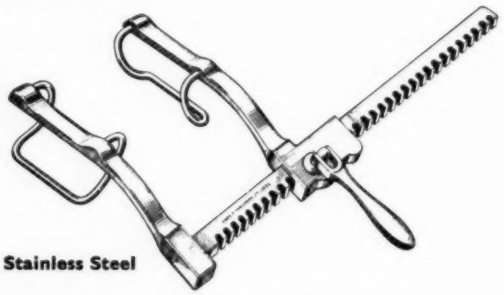

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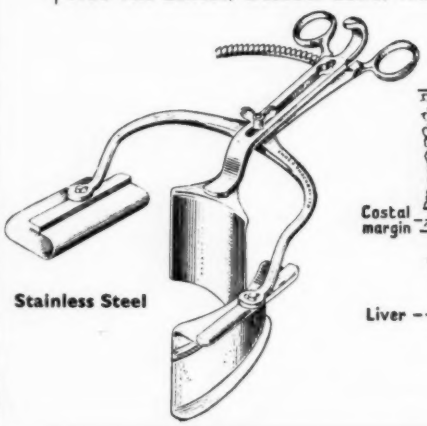

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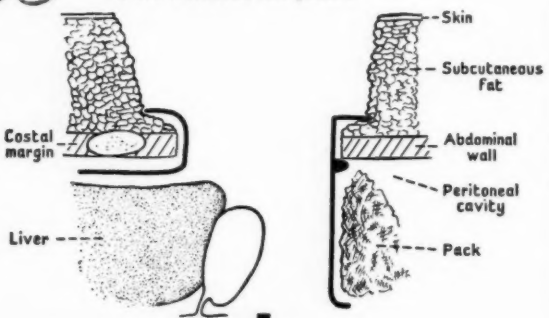

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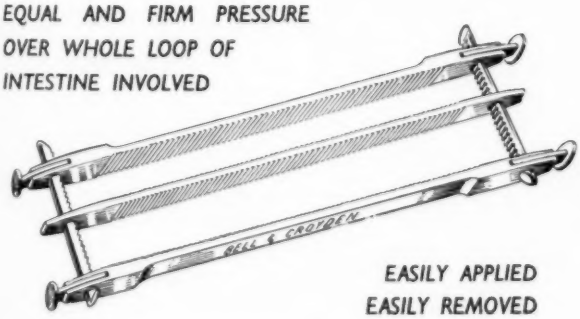

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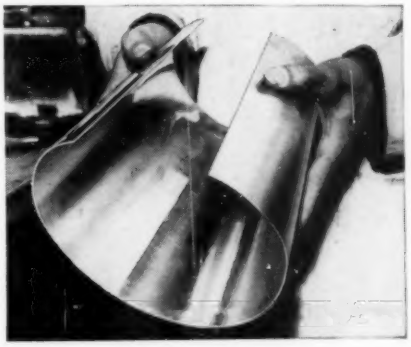

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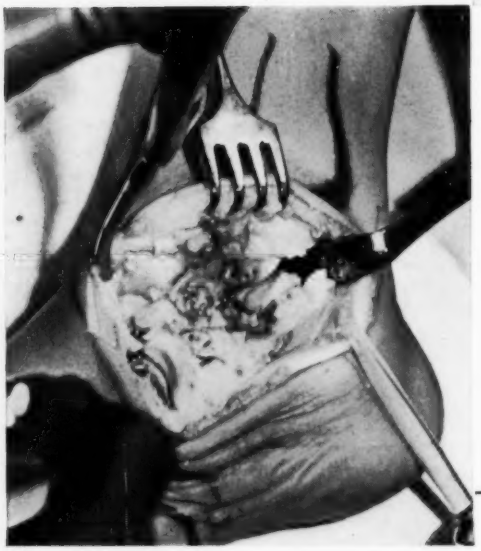

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